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VOLUME 20 NOVEMBER 1934 NUMBER 5

ROENTGEN CHANGES IN THE PETROUS PORTION OF THE TEMPORAL BONE WITHOUT CLINICAL MANIFESTATIONS

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Since Gradenigo first described his classic triad, numerous authors have endeavored to offer explanations for this condition. Careful histologic study has added much to the knowledge of disturbances of the temporal bone.

It is known that at least 80 per cent of the patients with Gradenigo's syndrome recover following simple mastoidectomy. In the 20 per cent who succumb, histopathologic study reveals that suppuration of the apex of the petrous portion of the temporal bone is more frequent than has been suspected.

It has been argued by many that Gradenigo's syndrome is not a distinct clinical entity, owing to the lack of adequate anatomic and pathologic bases. Numerous attempts have been made to show that palsy of the sixth nerve is due to involvement in Dorello's canal (Vail [1] and others), to venous congestion (Freisner and Druss,[2] Ruskin,[3] and others) or to localized meningeal irritation (Eagleton [4]).

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fifth Annual Session of the American Medical Association, Cleveland, June 14, 1934.

1. Vail, H. H.: Vidian Neuralgia with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Apex, Arch. Otolaryng. **17**:212 (Feb.) 1933.

2. Freisner, I., and Druss, J. G.: Osteitis of the Petrous Pyramid of the Temporal Bone Associated with Paralysis of the External Rectus, Arch. Otolaryng. **12**:342 (Sept.) 1930.

3. Ruskin, S. L.: The Venous Circulation of the Petrous Bone, and Its Clinical Significance, Ann. Otol., Rhin. & Laryng. **42**:961 (Dec.) 1933.

4. Eagleton, W. P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex, Arch. Otolaryng. **13**:386 (March) 1931.

We recently stated [5] that the syndrome of Gradenigo may be due to a disturbance in the venous circulation of the temporal bone, with resultant congestion. This, perhaps, results from venous anomalies or from anatomic differences in the sizes of the lateral sinus and jugular foramina.

Among many, there is still the popular misunderstanding that Gradenigo's syndrome and involvement of the petron [6] are synonymous. It has been shown that involvement of the sixth nerve is not diagnostic of petrositis. In fact, Kopetzky and Almour stated "that the presence of sixth nerve palsy is more apt to be against the diagnosis of petrositis." [7] During the course of this paper we shall describe a case (G. N., no. 8), in which Gradenigo's syndrome was present on the left side without petrositis, and in which the roentgenogram showed involvement of the right petron without Gradenigo's syndrome.

Druss stated:

> Although infections of the petrous pyramid and its apex may vary in intensity, not all infections in these structures include the fifth and sixth nerves. Cases have been reported in which a localized abscess was present at the apex without an involvement of the fifth and sixth nerves.[8]

The excellent monograph of Kopetzky and Almour [7] has done much to clarify the situation relative to the diagnosis of empyema or osteitis of the petron. They described a surgical attack for the treatment of this dangerous complication.

There are several types of involvement of the petrous pyramid, i. e.: (1) the type so well described by Eagleton,[9] osteomyelitis of the apex of the petron, occurring in the nonpneumatized or marrow-containing petron; (2) empyema of the apex of the petron, or osteitis as described by Kopetzky and Almour and occurring in the pneumatic temporal bone or in the coalescent type of mastoid, and (3) petrositis (Profant [10]), or involvement of the petron producing the Gradenigo syndrome.

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5. Ersner, M. S., and Myers, D.: An Aid to the Diagnosis of Intracranial Complications Resulting from Venous Circulatory Disturbance of the Temporal Bone, Offered by X-Ray of Lateral Sinus and Jugular Foramina, Laryngoscope **43**:800 (Oct.) 1933.

6. Petron in the following discussion is used to designate the apex of the petrous portion of the temporal bone.

7. Kopetzky, S., and Almour, R.: Suppuration of the Petrous Pyramid: Pathology, Symptomatology and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **40**:396 (June); 922 (Sept.) 1931.

8. Druss, J. G.: Anatomy and Pathology of Petrositis, Laryngoscope **41**:394 (June) 1931.

9. Eagelton, W. P.: (*a*) Localized Bulbar Cisternal Meningitis, Facial Pain and Sixth Nerve Palsy, and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:386 (March) 1930; (*b*) footnote 4.

10. Profant, H. J.: Gradenigo's Syndrome with a Consideration of "Petrositis," Arch. Otolaryng. **13**:347 (March) 1931.

Following the original reports of Kopetzky and Almour,[7] Profant,[10] Eagleton [11] and others in 1929, numerous communications appeared in the literature describing cases of petrositis in which recovery followed difficult surgical procedures. Recently, cures were reported following different operative procedures as suggested by Smith,[11a] Seydell,[12] Lillie and Williams [13] and others.

The cases reported in the literature have been those in which recovery followed a difficult surgical procedure, or in which death occurred from meningeal or intracranial complications, and the diagnosis of petrosal involvement was made or confirmed by autopsy.

Since (1932), we have followed the routine of making roentgenograms of the temporal bone, which included pictures of the lateral sinuses and jugular foramina, the significance of which we described in a previous communication,[5] and pictures of the petrous pyramids. These roentgenograms are used as key films for future studies or for comparison with subsequent films, in the event of the occurrence of complications necessitating the making of another roentgenogram. This is in accordance with the practice of Taylor [14] and Kopetzky.[15] In the course of these routine studies, we collected a series of cases in which the roentgenograms showed involvement of the apex of the petrous portion of the temporal bone. In all these cases recovery followed an extensive simple mastoidectomy, which gave adequate drainage. In one case (H. W., no. 7) changes in the petron were seen on roentgenographic examination, and recovery followed several myringotomies.

Taylor [16] has shown roentgenograms similar to ours, in which changes in the petrous apex occurred during the course of mastoiditis. Our observations were made independently and were in our records previous to the appearance of Taylor's report in the literature. Due credit must be given him for the pioneer work.

Other cases described in the literature were late cases which presented symptoms of meningeal irritation as did Eagleton's or, like those

11. Eagleton (footnotes 4 and 10a).

11a. Smith, C. H.: Infections of the Petrous Pyramid, West Virginia M. J. **28**:7 (Jan.) 1932.

12. Seydell, E. M.: Surgical Treatment of Suppuration of the Petrosal Pyramid, Ann. Otol., Rhin. & Laryng. **42**:455 (June) 1933.

13. Lillie, H. I., and Williams, H. I.: Suppuration of the Petrous Pyramid, Arch. Otolaryng. **15**:692 (May) 1932.

14. Taylor, H. F.: The Roentgen Findings in Suppuration of the Petrous Apex, Ann. Otol., Rhin. & Laryng. **40**:367 (June) 1931.

15. Kopetzky, S. J.: Otitic Complications as They Occur in Every Day Otology, Pennsylvania M. J. **36**:739 (July) 1933.

16. Taylor, H. K.: Suppuration of the Petrosal Pyramid: Roentgenologic Problems, Arch. Otolaryng. **19**:458 (Oct.) 1933.

of Kopetzky and Almour, showed the typical symptoms of ocular pain, otitic sepsis, etc.

We believe the findings herein reported to be important observations. They cannot be emphasized too frequently. Many on observing such roentgenograms and case histories as we are presenting would be tempted, no doubt, to operate immediately on the petron, especially if key plates had not been made before the operation. By conservative surgical treatment, recovery resulted in all these cases following extensive simple mastoidectomy, and in one case following myringotomy only. All of these cases occurred in patients with well pneumatized mastoids, which we can understand, in accordance with Wittmaack's theory of pneumatization.

It may be stated, without fear of contradiction, that in the past spontaneous recovery occurred in many cases of what is called petrositis, or recovery was aided by myringotomy or mastoidectomy, and the condition consequently remained unrecognized. This was because we were unaware that such a condition might exist. At the present time, by the use of key plates made prior to operation, we may have a graphic record of the condition of the petrous portion of the temporal bone. If, as in some of our own cases, changes are noted, we can study them by repeated roentgenograms. If there are no clinical signs we must wait and be strictly conservative in our attitude. If clinical signs and symptoms develop in addition to the roentgenographic evidence we are masters of the situation and can perform the indicated operation at any time. It must be reiterated that the decision to operate on the petrous apex must not rest on the roentgenographic picture. However, in the experience garnered in studying the cases that are to be reported, even in several which showed clinical signs suggesting pyramidal suppuration and in which the roentgenographic findings were positive, we still waited, especially when a well pneumatized mastoid was present. The condition in these cases cleared up without extensive radical operation. We emphasize this because the difficult surgical procedures may not be necessary as often as we now imagine, especially when the fear of a newly described condition wears off.

In support of this, we should like to quote Lillie and Williams: [18]

> It has been found by one of us (Dr. Lillie) that extension of the air cells into the petrous pyramid is occasionally encountered when the cells have been involved in disease of the mastoid without producing the symptoms so clearly described by Eagleton. In following the original recommendations of Küster, in which he advised the surgeon "to open up the bone freely so that it can be readily scrutinized to clear away all disease and so fully to expose the source of suppuration that the pus is nowhere checked in its outflow," such extensions are occasionally encountered, and the pyramid has been drained without knowledge of the specific symptoms of suppuration of the petrous pyramid.

In accordance with Wittmaack's theory, if there is no disturbance in the process of pneumatization, a well pneumatized mastoid will also show pneumatization of other portions of the temporal bone. This will result, in numerous cases, in pneumatization of the petrous portion of the temporal bone. We quote from Druss,[8] who mentioned the studies of Zuckerkandl, Belinoff and Balan:

> Just as the mastoid process may vary in its structure so may the petrous pyramid and its apex vary. Zuckerkandl, a good many years ago, studied anatomically 250 temporal bones. He found that the mastoid process was pneumatic in 36.8 per cent, diploetic in 20 per cent and mixed in 43.2 per cent. Recently Belinoff and Balan, of Bulgaria, studied grossly the petrous pyramids of 40 consecutive cases, particular attention being given to the anatomic structure of the apex. They found the apex pneumatic in 35 per cent, diploetic in 22.5 per cent and mixed in 42.5 per cent. In comparing these two sets of figures, we find that the per cent of each type is practically the same for the mastoid as for the apex. . . . On the contrary, in only 37.5 per cent of the cases was the structure of the same character in the mastoid as it was in the apex of the same temporal bone: Their structure varied in 62.5 per cent of the cases.

Druss further stated:

> While it is the rule for the mastoid process to be more pneumatic than the petrous pyramid and the apex, nevertheless the reverse condition sometimes exists, namely, the mastoid process is diploetic, while the paralabyrinthine cells and the cells at the apex are pneumatic. At times there may be as few as one or two cells at the apex. These anatomic variations, as will subsequently be pointed out, undoubtedly play an important role in the spread of infection from the middle ear to the apex.

The reason for the aforementioned variation in structure between the mastoid process and the petrous apex is perhaps due to the fact that if infection occurs in the temporal bone after pneumatization has occurred the parts involved may undergo sclerosis, whereas the more distal part goes on and becomes pneumatized. If infection occurs during the course of pneumatization, some portions of temporal bone may remain diploic and parts that escaped the infective process become pneumatized. We illustrate this by a case (H. B.) in which mastoidectomy was performed on the patient at the age of 1 year. Seven years later, a secondary operation on the mastoid revealed new bone in the mastoid process, while the apex of the petrous portion of the temporal bone was well pneumatized, as shown by a roentgenogram.

Our purpose in showing these statistics is to demonstrate that as more and more routine studies of the petrous apex are made more cases of this nature will come to light, and it will be seen that in pneumatized mastoids involvement of the petron is frequent and that in those cases it often resolves if an adequate mastoidectomy is performed. This may be compared to the exudative mastoiditis that occurs during acute otitis media.

Whether this is extension of suppuration by continuity or contiguity, or whether it is a congestive process due to venostasis (the occurrence of which we suggested) in cases that involve the side on which the larger venous structures are located is not, as yet, proved. But we believe that as time goes on the prognostic importance of the size of the lateral sinuses and jugular foramina and other vascular anomalies will be more and more appreciated.

There are two problems in dealing with a pneumatized mastoid: 1. A good chance for drainage is present in the large cellular type of mastoid bone. The edema of the mucous membrane does not reach a point where intracellular and intercellular drainage is impeded, and in these cases we can afford to be conservative even before an operation on the mastoid is resorted to. In this type of case absorption of bone occurs, and while there is more pus, better drainage is present. 2. In the cellular type, in which the cells occur in all directions, some distant or distal cell may be involved. If involvement occurs in the petron, and the edema and congestion are so great that the cell cannot drain, trouble begins. Analogous to this is the case in which the cells of the tip of the mastoid are involved, while the rest of the mastoid process remains almost normal.

We hope that with this preliminary explanation we have made clear our position. We shall attempt to illustrate further as the discussion of each case proceeds. The importance of this condition is fully realized by the members of this body, and we believe that this work presents it slightly differently from the accustomed fashion. In other specialties of medicine, as well as in our own, we are often tempted to follow classic procedures with a dogmatic blindness. We hope that this may help to make us more liberal. We salute our predecessors.

REPORT OF CASES

CASE 1.—*History.*—Mrs. C. McG., white, married, aged 20, was admitted to the Temple University Hospital, otologic service, on Feb. 4, 1933, with complaints of a painful discharging right ear, headache, fever and pain in the right eye. The present illness dated back to January 26, nine days before admission, at which time the patient was in the maternity ward of the Temple University Hospital. She had complained of a pain in the right ear, and on examination a diagnosis of acute suppurative otitis media was made, and myringotomy was performed. The ear continued to drain, but the condition improved so that the patient was discharged from the hospital and referred to the otologic outpatient department on January 30, four days after myringotomy. Soon after she left the hospital she began to complain of severe headache, pain in and behind the right ear and deep pain in the right eye. On February 4, nine days after the onset of the illness, she was readmitted to the hospital. The temperature was 100 F.

The past medical history had no bearing on the present illness.

Examinations.—The patient appeared to be fairly well nourished. The pupils reacted well and promptly to light and in accommodation. The extra-ocular movements were good and normal in all directions. There was no evidence of ocular

palsy. The mucous membrane of the nose was red and congested, with septal deflection to the left. The tongue was coated; the upper incisors had been extracted. The patient was unable to open the mouth far owing to pain over the right temporomandibular region. The tonsils had been removed.

There was postcervical and submaxillary adenopathy on the left. The left ear was normal. The right ear showed a profuse purulent discharge from the external auditory meatus. There was edema of the posterior superior wall of the canal with narrowing of the canal. Externally, there were redness and swelling over the area of the right mastoid, with periosteal thickening and pain on pressure over the tip of the mastoid and antrum.

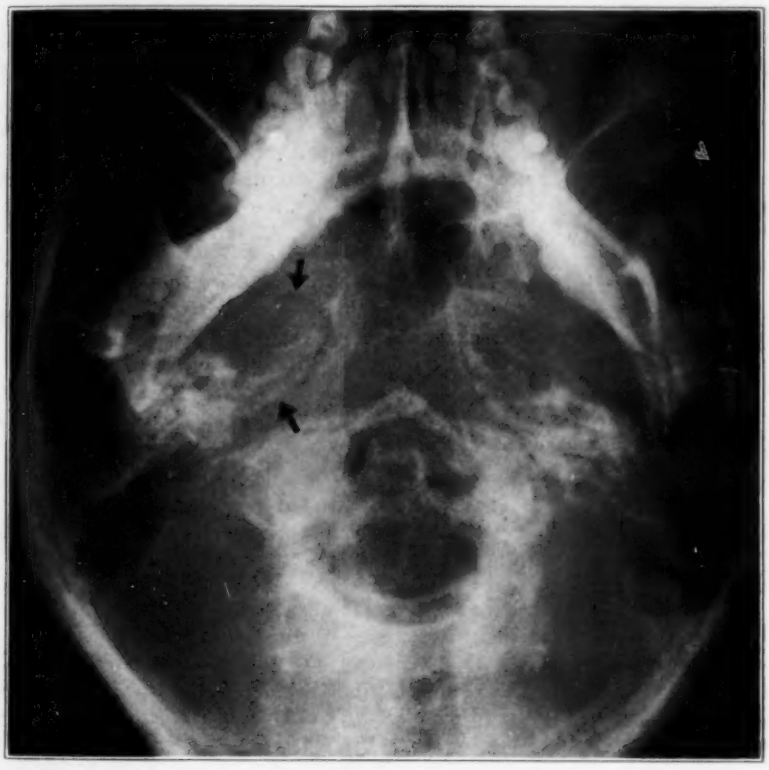

Fig. 1 (case 1).—The cellular structure in the mesial cephalic portion of the right petron is destroyed; the normal left side shows pneumatization.

Physical examination of other parts of the body gave essentially negative results.

Roentgenographic examination of the mastoids showed the following (Dr. A. K. Merchant): The cells of the left mastoid were normal. On the right side, all of the mastoid cells were filled with an abnormal density. These findings were well demonstrated. In the usual lateral stereoscopic projections of the two mastoids and in the roentgenograms, there was no definite evidence of destruction of the bone in the right mastoid. However, in addition to these studies, we obtained stereoscopic vertical projections of the base of the skull for the purpose of demonstrating the mesial portions of the petrons and jugular foramina. These films (fig. 1) showed some startling and interesting changes. A comparison of the

two petrons showed that there were cells to the extreme mesial end of the left petron, and the structure of the bone was entirely normal. On the right side, the cellular structure in the mesial cephalic portion of the petron was destroyed, and there was definite evidence of destruction of the bone in this region. The area of destruction was irregularly oval and measured about 1.5 cm. in length and 1 cm. in its widest dimension. The margins of this area of destruction were clearly defined. This may even be considered as an abscess in the right petron in the location described.

It should be pointed out that the left lateral sinus was larger than the right, and the left jugular foramen was correspondingly larger than the right.

On clinical examination, the following diagnosis was made: acute purulent otitis media and acute surgical mastoiditis. The roentgenographic examination was made just preceding the operation, so that the operation (since all clinical signs pointed toward mastoiditis) was begun before the films were read.

Operation.—This was our first case of this nature, and the results obtained by the simple operation on the mastoid without knowledge of the state of the petrous portion of the temporal bone led us to adopt this conservative attitude, which we continued to pursue. This was especially true since in the case of R. K., which had preceded this and presented similar symptoms, the Kopetzky-Almour operation failed to reveal pus on drilling the apex.

At operation, a mastoid of the large cell type was found, presenting a coalescent mastoiditis, with considerable necrosis of cellular septums and pus. These were completely exenterated. The cell destruction extended well into the zygomatic region, which was thoroughly cleaned out. The lateral sinus was exposed as far as the jugular bulb. The patient's immediate postoperative condition was good. At the close of the operation, we became aware of the roentgenographic findings as detailed previously. A thorough mastoidectomy having been done, it was decided to observe the patient carefully, before any further surgical procedures were considered.

Course.—On the second postoperative day the temperature was 100 F.; on February 13, the fourth postoperative day, there were swelling over the right temporal region and zygoma. There was difficulty in opening the jaw and on swallowing and mastication.

On February 16, seven days after operation, the temperature was normal and the mastoid wound was healing nicely, but there was a slight discharge from the external auditory meatus. The patient still complained severely of pain in the right eye, which seemed to be retro-orbital and worse at night, requiring sedation. There was no evidence of any ocular palsy. On February 22, thirteen days after operation, there was still considerable drainage from the middle ear. The pain in the eye, although still present, had abated considerably.

On March 6, twenty-five days after operation, the temperature was normal, the middle ear was dry, and the orbital pain had practically disappeared. A roentgenogram at this time showed practically no change; however, there was no progression of the involvement of the petron, which was interpreted as meaning that the condition was subsiding in view of the adequate drainage provided.

March 13, thirty-two days after operation, the temperature was normal. No neurologic signs were present. The mastoid wound was well healed, and the middle ear was dry. The patient was discharged and referred to the otologic outpatient department. Follow-up examination in the dispensary, the patient being seen twice weekly for more than a month, showed no recurrence, and the patient was discharged. One year later the condition was still good, without evidence of recurrence.

Examinations made on February 13, 17 and 24 showed the eyegrounds to be normal, with no other disturbance of ocular function.

Summary.—The following points summarize the case: (1) acute suppurative otitis media on the right, with severe pain in the ear; (2) pain in the right eye, retro-orbital, nocturnal and persistent; (3) low grade sepsis; (4) acute suppurative mastoiditis; (5) well pneumatized extensive mastoiditis; (6) roentgenographic evidence of involvement of the petrous apex on the right side; (7) extensive simple mastoidectomy; (8) persistence of ocular pain and aural discharge following operation; (9) a large lateral sinus and jugular foramina on the left side, which provided adequate venous drainage, and (10) recovery of the patient without further surgical procedure.

Comment.—We believe that this was a definite case of petrositis (the type described by Kopetzky and Almour). Although the clinical course did not closely follow their classic description, there was the presence of the aural discharge and ocular pain preoperatively and postoperatively, with mild sepsis and a questionable quiescent period.

There is a question as to whether the involvement of the petron was suppurative or congestive. To date this cannot be differentiated by roentgenographic examination. However, the fact that the roentgenogram of the petron showed some cell destruction indicated that it was probably a true petrositis or osteitis.

We believe that drainage was facilitated by the thorough simple mastoidectomy. The fact that the mastoid was extensively pneumatized allowed for avenues of drainage, and the presence of the large lateral sinus and jugular foramen on the left allowed for proper contralateral circulation. This will be discussed more thoroughly later.

CASE 2.—*History.*—W. S., a man, white, aged 21, was seen in the otologic dispensary of the Temple University Hospital, with a history of pain in the right ear and aural discharge, following spontaneous perforation. The ear had been draining for the past two months. At the time the patient was seen in the dispensary, the temperature was 99.4 F.; there was profuse drainage from the middle ear with definite clinical evidence of mastoiditis. The patient was referred to the ward of the Temple University Hospital for operation.

Examinations.—On admission on March 23, 1933, two months after the onset of the symptoms, the temperature was 99.4 F. There was profuse purulent discharge from the right external auditory canal, and on otoscopic examination granulation tissue and a small polyp were seen on the right ear drum. There was some edema of the posterior superior wall of the canal and there were periosteal thickening and tenderness over the mastoid.

Roentgenographic examination gave the following results (Dr. A. K. Merchant): Both mastoids were normally developed and pneumatized. The cells of the left mastoid appeared normal. Abnormal density filled all of the mastoid cells on the right side. The vertex projections (fig. 2) showed a large oval area of bone destruction in the mesial end of the right petron. The jugular foramina were practically equal in size, both of them being within normal limits. The right lateral sinus appeared to be slightly larger than the left. There was a rather large

emissary vein in each mastoid. It was concluded that there was acute mastoiditis on the right side, with evidence of bone destruction in the mesial end of the right petron, as previously described.

Operation.—Prior to operation a diagnosis of subacute suppurative otitis media and acute mastoiditis on the right side, with involvement of the right petrous apex, shown roentgenographically, was made. At operation, performed on March 23, the day following admission, a considerable amount of pus and cellular necrosis were found in the mastoid bone. This was thoroughly exenterated. The lateral sinus was exposed near the sigmoid and close to the jugular bulb, and infected

Fig. 2 (case 2).—Note the large oval area of bone destruction in the mesial end of the right petron. The left side is normal.

cells in the region were removed, as were the peribulbar cells. The tip was greatly necrosed, and the cells were removed almost to the styloid process and into the region of the pterygoid bone. The posterior bony canal was trimmed down in the manner of a modified Heath operation. Cells over the dural plate were removed with the exposure of an epidural abscess, from which about 2 cc. of pus escaped. Cells in the region of the zygoma were thoroughly removed. The wound was irrigated with saline solution and packed with iodoform gauze, and the skin was closed with interrupted dermal sutures. The immediate postoperative condition was good.

Course.—The mastoid wound and the middle ear healed rapidly and remained dry. There was no complaint of ocular pain; the eyegrounds were normal. The

patient was discharged on April 7, fourteen days after operation, in good condition. Follow-up study in the otologic clinic for one month revealed no change in the condition.

Summary.—The following points summarize the case: (1) a discharging right ear for the past two months; (2) extensively pneumatized mastoid; (3) roentgenographic evidence of petrositis; (4) extensive mastoidectomy; (5) absence of clinical signs of petrositis, and (6) recovery without complication following simple mastoidectomy.

Comment.—The patient presented an extensively pneumatized temporal bone with cells extending to the region of the styloid process, peribulbar cells, perilabyrinthine cells and subarcuatic cells. In removing all these infected cells, we promoted good drainage. In addition, there were adequate venous structures, and the patient went on to an uneventful recovery. It might also be added that the presence of the epidural abscess may be taken as an indication that the infection emptied itself in this manner.

Case 3.—*History.*—Miss M. W., white, aged 20, was admitted to the private service of Dr. M. S. Ersner on March 28, 1933. Ten days before a sore throat had developed and eight days before, a pain in the right ear. The ear drum was incised and had drained since. There was considerable pain in the right ear, the right side of the neck and behind the ear. Two days before admission to the hospital, the ear drum was reincised. The temperature on admission was 102.3 F. The past history was unimportant except for a constant postnasal drip, which the patient had for some time.

Examinations.—The left ear was normal. There was a profuse greenish discharge from the external auditory canal of the right ear. The ear drum was under tension and pulsating. There were narrowing of the external auditory canal and sagging of the posterior superior wall of the canal. At the time of the examination, the patient had a slight chill and was perspiring freely.

There was absence of neurologic signs. Other systems of the body were normal on physical examination.

Roentgenographic examination by Dr. W. E. Chamberlain was reported as follows: The appearance of the left mastoid and the left petron was normal. Both mastoids were rather extensively pneumatized with well developed air cells in the tip of the petron and in the mastoid process and petrous pyramid.

On the right side, there was extensive exudative mastoiditis, as a result of which the air was excluded from all of the cells of the mastoid and at the tip of the petron and of the petrous pyramid (fig. 3). In addition to this evidence of exudative mastoiditis, it was thought that there was an actual breaking down of bony septums throughout the tip of the mastoid process, where many of the bony septums are absent. Dr. Merchant and Dr. Chamberlain considered the possibility that this might be a matter of anomalous development, but decided that they were dealing with bone suppuration and destruction.

The jugular foramina was also studied, the one on the right side being extremely large, while the one on the left was of rather moderate size but not abnormally small. Conforming to this finding, the impression on the inner table of the skull of the right lateral sinus was much larger than the corresponding impression of the left lateral sinus.

It was concluded that there was mastoiditis on the right side with involvement of the tip of the petron as well as other parts, and what was believed to be a suppurative breakdown of the bony septums in the tip of the mastoid process.

The diagnosis at this time was acute suppurative mastoiditis on the right side and involvement of the right petron, shown roentgenographically.

Operation.—On March 28, under nitrous oxide-ether anesthesia, an extensive simple mastoidectomy was done on the right side. There was a thick cortex, on removal of which a large abscess cavity was located. Pus, granulation tissue and necrotic

Fig. 3 (case 3).—Note the involvement of the right petron, with the exclusion of air from all the cells. On the left side, cellular septums are distinctly visible.

cells were exenterated. The lateral sinus and dura of the middle fossa were exposed and found to be normal. The cells in the zygomatic region were involved and were thoroughly cleaned out. The postoperative course was uneventful, and the patient was discharged on April 10, twelve days after admission, with a dry middle ear and a healing mastoid wound. Follow-up treatment revealed no change in the patient's condition, and to date there has been no recurrence of symptoms.

Summary.—The following points summarize the case: (1) tonsillitis followed by otitis media; (2) acute suppurative otitis media on the right side; (3) two myringotomies on the right side; (4) acute suppurative mastoiditis on the right,

coalescent type; (5) involvement of the right petron, as demonstrated by the roentgenogram; (6) extensive simple mastoidectomy, and (7) an uneventful postoperative course.

Comment.—This mastoid was of the large cellular type and on roentgenographic examination there was shown involvement of the right petron. Whether this was a suppurative process or a congestive process cannot be differentiated at present by roentgenographic examination. Nevertheless, in a pneumatic mastoid of this type there are definite pathways of cells leading to the petron. By promoting adequate drainage, resolution occurred.

At no time did the patient present clinical symptoms of petrositis. In this case the involvement of the petron completely resolved. It was noticed that there is an extremely large lateral sinus and jugular foramina on the right side, while on the left side these structures were much smaller, yet within normal limits. It can be readily seen, therefore, that an acute inflammatory process involving the right temporal bone could easily produce venous stasis and congestive phenomena, which would produce the changes in the petron that we noticed in the roentgenogram. The saving factor, in addition to the operation, was that the blood vessels of the left side, although much smaller in size, were able to care for the disturbed circulation.

CASE 4.—*History.*—R. K., a man, white, aged 39, was admitted to the Temple University Hospital on March 30, 1932. The complaints on admission were discharge and pain in the right ear. The present condition dated back to three weeks before admission, at which time the patient had impacted cerumen washed from the right ear. Following this, he stated that he had a stuffy feeling in the right ear, and soon after the ear began to drain. The ear drum was incised to insure better drainage. The patient had grip at the time of the onset of the symptoms.

Examinations.—Physical examination gave negative results except for the right ear, which showed a profuse discharge from the external auditory canal. The ear drum was red and bulging, and a large perforation was visible. There were narrowing of the external auditory canal, sagging of the posterior superior wall of the canal and three point mastoid tenderness.

Operation.—The diagnosis at this time was acute suppurative otitis media and acute mastoiditis on the right side. Owing to the positive clinical findings, no roentgenogram was made at this time. On March 30, the day of admission, a simple mastoidectomy was performed. A necrotic purulent mastoid was found. The dura of the middle fossa was exposed.

Course.—Following the operation, the patient's temperature remained elevated. There was no nuchal rigidity or facial palsy. The patient was coughing somewhat, but physical examination of the chest gave negative results. On April 2, three days after operation, the patient's temperature was 104 F. There were no meningeal signs. The cough persisted, and a few scattered râles were heard through the chest.

On April 7, the eighth postoperative day, the temperature dropped to normal and then rose to 103 F. by 11 a. m. There was considerable drainage from the mastoid wound, and the patient had several chills. Sinus thrombosis was suspected,

although no definite signs were present. A blood culture taken on this day was later found to be negative. On April 8, the ninth postoperative day, the temperature was still of the septic type, rising to 104 F. daily. An Ayer-Tobey test gave negative results. The spinal fluid was normal. The patient was given daily intravenous injections of 10 cc. of 0.5 per cent acriflavine base. The temperature gradually dropped to normal, and on April 13 the patient was discharged and referred for follow-up treatment. While at home he began to complain of severe headaches and diplopia, and was readmitted to the otologic ward service on April

Fig. 4 (case 4).—Note the changes in the apex of the right petron.

19, twenty days after operation. The complaints on admission were severe headaches, diplopia and retro-orbital pain. On the day following his discharge from the hospital, the patient had recurrent attacks of severe headaches and pain over the right eye and right temple, which were sufficiently severe to keep him awake all during the night. In the morning he felt better, so that he drove to the doctor's office, but on returning noted that he was seeing double. His appetite was poor. He was frequently nauseated, but never vomited.

Examinations.—On April 19, the temperature was 98 F.; the pulse rate, 75 per minute, and the respiratory rate, 20 per minute. The pupils were dilated from a mydriatic. There was definite weakness of the right external rectus muscle. No nystagmus was present. The left ear was normal. The right mastoid wound was

still showing a thick creamy discharge, and there was considerable discharge from the right external auditory canal. There was no facial weakness or other neurologic phenomena.

Roentgenographic examination by Dr. W. E. Chamberlain on April 18 was reported as follows: Pictures were made of a number of special angles of the skull, which were difficult to interpret, but on the right side, in the region of the suture between the basilar process of the occipital bone and the tip of the petrous portion of the temporal bone, there was definite absence of bone. The bone destruction or absence of bone did not appear to affect the occipital or the sphenoid bones but definitely involved the tip of the petron. It should be borne in mind that the condition, as visualized in these roentgenograms, might possibly be a developmental asymmetry of the petron, but the important possibility, which is borne out to some extent by the clinical findings, is that of a suppuration of the bone in this part of the right petron (fig. 4).

The ocular examination, by Dr. Boehringer, showed some fulness of the retinal vessels in the right eye; the optic disks were well defined, and the left eye was normal.

The Bárány test, performed by Dr. J. Winston, showed an appreciable loss of function of the vestibular apparatus on the right. The hearing on this side was considerably impaired. There was more marked loss of function of the left vertical semicircular canals, while the left horizontal canals responded fairly well. The hearing was good in the left ear. These findings were suggestive of intracranial disease. The cerebellum, cerebellopontile angle and posterior cranial fossa appeared to be normal. The lesion was probably extradural and in the right parietal region.

The diagnosis at this time was: (1) Gradenigo's syndrome and (2) petrositis.

Operation.—In view of the findings of involvement of the right petron, it was decided to reopen the mastoid and to perform the Kopetzky-Almour operation for drainage of the apex of the petrous pyramid. This was done on April 25, twenty-six days after the first operation. There was no free pus when the pyramid was entered. A decompression of the middle fossa through the tegmen tympani was done, and exploration for pus at the base of the brain was made. None was found. Following the operation, the patient made an uneventful recovery; the weakness of the lateral rectus muscle disappeared by the time the patient was discharged on April 29, ten days after his second admission and thirty days after the original mastoidectomy.

On follow-up examination it was found that the patient did well, and that there had been no recurrence of symptoms.

Summary.—A summary of the main points of the case follows: (1) acute otitis media on the right, and myringotomy; (2) mastoidectomy on the right on March 30, three weeks after the onset of the illness; (3) a stormy postoperative course, with a septic type of temperature, a curve suggestive of sinus thrombosis, and a negative Ayer-Tobey test and blood culture; (4) discharge from the hospital on April 13; (5) occurrence of pain in the eye, headache and aural discharge on April 17; (6) palsy of the right external rectus muscle and diplopia on April 19; (7) roentgenographic evidence of petrositis, and (8) a Kopetzky-Almour operation, with recovery and discharge on April 29.

Comment.—This is a case in which symptoms typical of Gradenigo's syndrome developed, and it might be said to be somewhat typical of petrositis, and yet at operation no pus was found on tapping the petron, although the roentgenogram showed involvement of the petron. In

view of our experience with these cases, we no doubt now could have been tempted to wait much longer before considering an operative procedure. However, this was a valuable case in view of the fact that it demonstrated that the involvement of the petron was probably congestive, and that in these large cellular mastoids adequate drainage produced by a thorough mastoidectomy may have resulted in resolution by providing avenues of drainage. We cannot say that cure was due to the Kopetzky-Almour operation, and the only harm done was the destruction of the patient's middle ear.

CASE 5.—*History.*—M. L., a man, white, aged 25, a lawyer, was admitted to Temple University Hospital, service of Dr. Matthew S. Ersner, on July 13, 1933. The chief complaints on admission were pain in the left ear, radiating to the occipital region, and profuse aural discharge. The present illness dated back to July 2, at which time the patient went swimming, and the next day severe pain developed in the left ear. On July 4, myringotomy was done. This was repeated on July 7. There was still profuse discharge from the left ear with considerable pain, although not so severe as at the time of onset.

Examinations.—Physical examination gave negative results, except for the left ear, which presented a profuse thick, greenish discharge. There were slight narrowing of the canal, drooping of the posterior superior wall of the canal and a large perforation in the posterior superior quadrant of the ear drum. The drum membrane was pulsating and under tension. On July 15, two days following admission, after consultation with Dr. G. M. Coates, another myringotomy was done under nitrous oxide anesthesia. This was followed by rather severe bleeding, necessitating packing of the canal. This soon was controlled, and the ear began to drain freely. The patient continued to complain of pain in the side of the face and eye, which was worse at night, so that he was unable to sleep and required numerous sedatives. On July 20, twenty days after the onset of the illness, no improvement having been manifested, and in view of the roentgenographic findings, it was decided to perform a simple mastoidectomy.

Roentgenographic examination, by Dr. Chamberlain, on July 13, showed both mastoids to be well developed and pneumatized. The cells of the right mastoid appeared clear throughout. On the left side, all the mastoid cells showed abnormal density. In the zygomatic portion of the left mastoid there was some destruction of cell outlines. The right jugular foramen and the right lateral sinus were larger than the corresponding structures on the left. Vertex projections of the petrous portions of the temporal bone showed some pneumatization of the right petrous tip. On the left side, there was abnormal density in this region (fig. 5).

It was concluded that there was exudative mastoiditis on the left side, involving the petrous tip. There was some break-down of cells in the zygomatic portion of the left mastoid. Another roentgenographic examination on July 17, for comparison showed no appreciable change in appearance.

The diagnosis was (1) acute purulent otitis media on the left; (2) acute mastoiditis on the left, and (3) roentgenographic evidence of involvement of the left petron.

Operation.—On July 20, under rectal and inhalation anesthesia, an extensive simple mastoidectomy was performed. Pus, necrosis and granulation tissue were exenterated. The lateral sinus and the dura of the middle fossa were exposed

and seemed to be normal. The cells extended well into the zygomatic region, which was thoroughly cleaned out, and into the occipital region, behind the lateral sinus; these were also removed.

Course.—Postoperatively, the condition remained good; the pain in the eye and the ear and the headaches disappeared. The middle ear became dry, and the mastoid wound healed rapidly, so that the patient was discharged on July 30, seventeen days after admission. Follow-up treatment revealed no change in the patient's condition. There has been no recurrence of symptoms up to the time of this report.

Fig. 5 (case 5).—Note the abnormal density in the region of the left petrous apex. Normal pneumatization is shown on the right side.

Summary.—The following points summarize the case: (1) pain in the ear, July 2; (2) left myringotomy, July 4; (3) repeated myringotomy, July 7; (4) repeated myringotomy, July 15; (5) acute mastoiditis; (6) roentgenographic evidence of involvement of the left petrous apex; (7) extensive simple mastoidectomy, July 20, and (8) uneventful recovery.

Comment.—This case showed roentgenographic evidence of petrositis, occurring in a well pneumatized mastoid. The cells extended to every part of the temporal bone. There were also clinical symptoms

of pyramidal involvement. Recovery ensued following the extensive simple mastoidectomy. The presence of larger venous structures on the right, providing adequate contralateral drainage aided considerably.

CASE 6.—*History.*—H. B., aged 8, a boy, white, was admitted to the otologic service of the Temple University Hospital on Jan. 25, 1934. Mastoidectomy had been performed on the left side at the age of 1 year. Since that time, the child had been free from complaints relative to this ear until two weeks previous to his admission to the hospital.

During August 1933, about five months previous to admission, while at the seashore, a cold developed, which became chronic. The child was treated for sinusitis, without much relief. Two weeks previous to his admission to the hospital pain developed in the right ear, with profuse purulent discharge. This failed to respond to local treatment of the ear, nose and throat. He was seen on the day of admission with a temperature of 101 F., postauricular redness, swelling, fluctuation and tenderness. A diagnosis of recurrent mastoiditis was made, and he was referred to the hospital for operation.

At operation, considerable pus, granulation tissue and necrotic cells were encountered. These were thoroughly exenterated. Following this, the child made an uneventful recovery and was discharged from the hospital in five days (Feb. 1, 1934). He was well, except for a slight amount of drainage from the mastoid wound, until February 8, eight days after operation, when he complained of being very tired and of a headache. This was relieved by acetylsalicylic acid and an enema. The following day, he again complained of headache and dizziness and pain in the left eye. This persisted for several more days. The pain was sufficiently intense to keep him awake during the night.

During this time, there had been an increase in the amount of discharge from the mastoid wound as well as a recurrence of drainage from the middle ear. The morning of readmission to the hospital, which was February 12 (eleven days after being sent home), he again complained of headache, dizziness and pain in the eye. During the past six weeks, he had suffered from anorexia, insomnia and loss of weight.

The past medical history, except for the mastoidectomy at the age of 1 year, is unimportant in this instance.

Examinations.—Physical examination revealed the following: There were no positive ocular changes. The pupils were equal and reacted to light and in accommodation; the media were clear; the fundi and extra-ocular movements were normal; the ocular fields were grossly normal (Lillie).

Neurologic examination (Nathan) revealed no involvement of the cranial nerves. There was no objective involvement of the fifth nerve. The reflexes were normal; there was no evidence of cerebellar dysfunction or focal neurologic signs.

The Bárány examination (Winston-Myers) was performed. The right, or good, ear was douched; the left ear was not douched because of the dressing. It was not thought advisable to turn the patient. The right vestibular apparatus functioned fairly well. The hearing was good in both ears. Clinically, the symptoms suggested some inflammatory lesion in the region of the left petrous pyramid. From a vestibular standpoint, the entire opposite vestibular mechanism should show impairment of function. As stated previously, the opposite vestibular mechanism responded fairly well.

The heart and lungs were normal.

Laboratory findings on the second admission were: hemoglobin, 13 Gm.; red blood cells, 4,200,000; white blood cells, 6,950; Schilling index, 3.2 $\left(\frac{45 \text{ mature}}{14 \text{ immature}}\right)$. The urinalysis gave negative results.

The roentgenographic examination, on February 2, by Dr. A. K. Merchant, revealed the following: The right mastoid presented a normal appearance. On the left side, two operations had been performed on the mastoid, but air cells could still be recognized. These cells were abnormally gray, but whether or not they were the site of active disease it was difficult to say. The most important finding

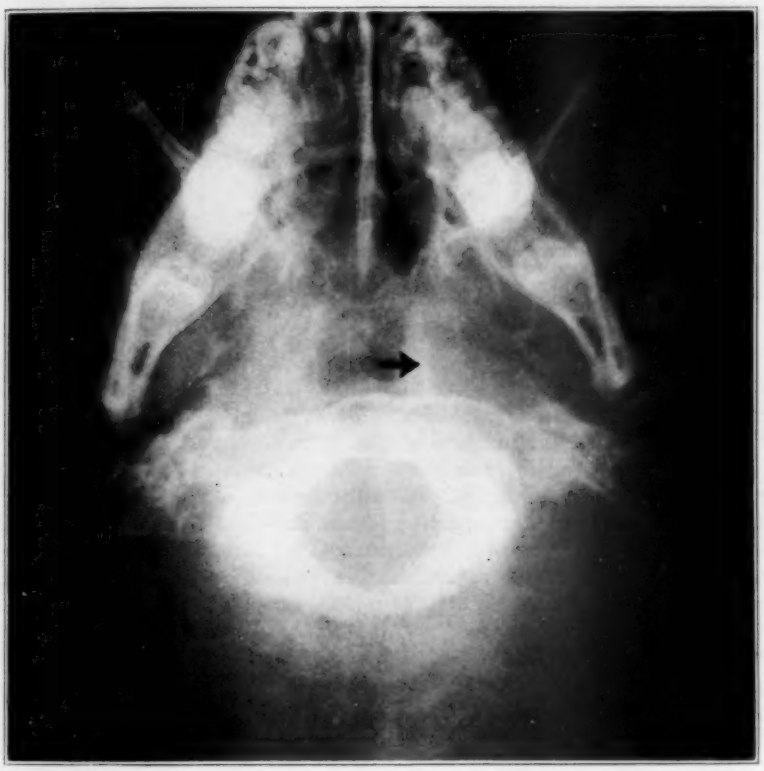

Fig. 6 (case 6).—Note the presence of irregular bone destruction in the left petron near its tip. Practically the whole petron shows irregular moth-eaten bone destruction, with what appears to be several tiny sequestrums.

was the presence of irregular bone destruction in the left petron near its tip. Practically the whole petron showed irregular, moth-eaten, destruction of the bone, with what appeared to be several tiny sequestrums (fig. 6). The right petron presented a normal roentgenographic appearance. It was concluded that there was petrositis on the left side, following mastoiditis of long standing.

Progress.—The patient had been admitted to the hospital with a probable diagnosis of petrositis. This was confirmed by roentgenographic examination. Other physical signs were negative. The child's general condition was good. It was decided to wait and observe him carefully before any radical procedure was

attempted. The wound was redressed daily. The temperature, pulse and respirations were recorded at intervals of two hours. The blood pressure was taken three times daily. The patient's temperature never reached higher than from 99 to 100 F. (rectal).

On February 18, six days after admission, the patient felt much better. The dizziness and ocular pain had practically disappeared. The middle ear was dry at this date. There was only a slight amount of drainage from the mastoid wound. Up to this time, radical operative procedure had been seriously considered. However, the marked improvement decided us in favor of waiting, not forgetting, however, that the patient might be in the so-called dangerous, quiescent stage.

Daily examination, daily dressings and roentgenographic examination failed to reveal any change in the child's condition. He did not complain any more of headache, dizziness or vomiting. He played about the ward, and his general condition was good. On February 28, sixteen days after admission to the hospital, he was discharged in good condition. The mastoid wound was well healed, and the middle ear was dry.

About one week after the patient's return home, scarlet fever developed. This produced an exacerbation of the otitic picture, and acute otitis media developed on the left side with a postauricular abscess. The ear drum and postauricular abscess were incised. With daily dressings at home, the condition cleared up. At no time during the course of the exanthem was there a recrudescence of symptoms suggesting pyramidal involvement.

Summary.—The following points summarize the case: (1) mastoidectomy at the age of 1 year; (2) sinusitis for five months previous to the onset of the aural condition; (3) acute suppurative otitis media on the left two weeks before admission to the hospital; (4) recurrent mastoiditis with postauricular abscess on February 25; (5) secondary mastoidectomy on February 25; (6) uneventful recovery; (7) onset of headaches, pain in the eye, dizziness and nausea eight days after return home; (8) symptoms for several days, and readmission to the hospital; (9) physical signs and a history suggestive of petrositis, confirmation by roentgenographic evidence of sequestration in the petron; (10) no other alarming signs on careful study and consultation; (11) draining mastoid and middle ear; (12) watchful waiting, good recovery, freedom from symptoms and discharge of the patient on February 28, sixteen days after the onset of symptoms, and (13) scarlet fever one week later, with postauricular abscess, but no return of symptoms suggesting petrositis.

Comment.—This case presents several interesting features. 1. There was a history of recurrent mastoiditis. At operation, we found new bone formation in the mastoid process, but the petrous apex was well pneumatized according to roentgenographic examination. This illustrates the point brought up early in the paper, that although a disturbance in pneumatization may result in a sclerotic or diploic mastoid, other portions of the temporal bone may go on to pneumatization. 2. The patient's symptoms were almost typical of Kopetzky and Almour's syndrome, viz., nocturnal orbital pain, dizziness, nausea, insomnia, headache and a draining middle ear and mastoid wound. The roentgenogram showed definite involvement of the left petrous apex; in fact, it even suggested sequestration. In view of the patient's good condition and the cellular distribution, it was decided to wait. This was

rewarded by the discharge of the patient from the hospital, well and without operation, in sixteen days.

The roentgenogram of the lateral sinuses and jugular foramina showed the structures on the right side to be larger, and the adequate circulation no doubt aided in resolution. The fact that scarlet fever developed after the patient returned home, and with it an exacerbation of the otitic picture, failed to stir up the process in the petrous pyramid.

CASE 7.—*History.*—H. W., a girl, aged 6, white, was admitted to the Temple University Hospital on March 2, 1934. The temperature was 100.2 F.; the pulse rate, 105 and the respiratory rate, 20. The chief complaints on admission were pain and a discharging right ear. The child was in good health until February 12 (eighteen days previous to admission), when she caught a common cold, which was accompanied by hoarseness and a slight fever. Fourteen days after the onset of the illness, the mother noticed that her right ear was draining. The patient was taken to an aurist, who incised her ear. The discharge persisted with pain, fever and tenderness over the mastoid. The child was referred to this hospital with the diagnosis of probable mastoiditis on the right side.

The patient had had measles at the age of 6 months, and simple mastoidectomy had been performed on the left side at the age of 3 years.

Examinations.—Physical examination gave essentially negative results, except for the aural pathologic process. The left ear was dry; there was a well healed postauricular scar of a previous mastoidectomy. The right ear showed profuse purulent drainage from the external auditory canal. The pus was thick, greenish yellow and mucoid. The right ear drum was nipple-shaped, under tension and pulsating.

Progress.—Immediately on admission, the right ear drum was reincised under ethyl chloride anesthesia. This was accompanied by free bleeding with escape of pus and the release of tension. Roentgenographic examination was made with the following report (Dr. A. K. Merchant): The left mastoid showed an operative defect. The peripheral cells of the left mastoid were missing, but the cells near the auditory canal were radiolucent and appeared entirely normal. On the right side, the mastoid was normally developed and pneumatized. All of the cells were filled with abnormal soft tissue density. There was no conclusive evidence of destruction in the right mastoid.

The vertex projection revealed some interesting findings in this case. The tip of the petrous portion of the left temporal bone was extensively pneumatized, all of the cells appearing clear. In the case of the petrous portion of the right temporal bone, the cells at the tip were abnormally gray, similar to the appearance of the mastoid cells of the right side (fig. 7).

The lateral sinuses and jugular foramina were large on both sides, those of the right side being a little larger than those of the left.

It was concluded that there was exudative mastoiditis on the right side, concomitant with which there was petrositis.

With these findings in mind, it was decided to keep the patient under careful observation, observing first the effect of the multiple myringotomies.

Laboratory findings at the time of admission were: hemoglobin, 10.5 Gm.; red blood cells, 4,410,000; white blood cells, 17,050; Schilling index, 3 $\left(\frac{63 \text{ mature}}{27 \text{ immature}}\right)$, and blood sedimentation index, 25 mm. drop in twenty-five minutes. The urinalysis gave normal results.

The eyegrounds were normal.

The patient was examined daily. The ear continued to drain profusely. The pain and tenderness over the mastoid disappeared. The temperature fluctuated daily, never rising over 100 F. (rectal). At the end of the first week in the hospital there was an abatement of the amount of drainage from the right ear; the ear drum began to retract, and the patient appeared better clinically. On March 15 (twelve days after admission), roentgenograms were again made of the mastoids and the apex of the petrous pyramids, and there was no appreciable change from the previous picture. On March 21 (eighteen days after admission),

Fig. 7 (case 7).—Note that in the petrous portion of the right temporal bone the cells at the tip are abnormally gray.

the ear was dry, the temperature was normal, and the patient was discharged from the hospital. Follow-up examination, made at weekly intervals, revealed no change in the patient's status.

Summary.—The following points summarize the case: (1) onset of acute otitis media on the right side, following a cold; (2) spontaneous perforation of the ear drum, with incision of the drum for better drainage; (3) onset of symptoms suggestive of mastoiditis; (4) myringotomy under general anesthesia, with release of pus and considerable bleeding; (5) roentgenographic evidence of involvement of the tip of the right petrous portion of the temporal bone, which was well

pneumatized; (6) relatively large size of the lateral sinuses and jugular foramina, the right being slightly larger than the left; (7) a dry ear and absolute recovery within three weeks following several myringotomies, and (8) no evidence of return of symptoms.

Comment.—This case presents several interesting findings. In accordance with our own studies, and corroborating those of Taylor, it is known that in a well pneumatized temporal bone during the course of acute otitis media, roentgenographic examination will show exudative changes in all portions of the temporal bone, especially if this happens to be well pneumatized, as in this particular case. The important thing, however, is that with all this extensive involvement, as elicited by roentgenographic examination, recovery took place following myringotomy. At no time were there symptoms of orbital pain, otitic sepsis, etc., such as those mentioned by Kopetzky. It was not necessary in this case to do a simple mastoidectomy, as has been our habit in other cases that showed findings of this nature. Another important finding is that the ear drum bled freely following the myringotomy, aiding in reducing congestion in the temporal bone. This has been described by us previously, as already quoted, and more recently by Ruskin.[3] The importance of an adequate venous circulation is again illustrated, this patient being especially fortunate in that both of the venous structures were of large size, especially in that the right side was larger than the left.

CASE 8.—*History.*—G. N., a boy, aged 8, was referred to the otologic service of Temple University Hospital by Dr. Oram Klein. On Jan. 7, 1934, the patient had scarlet fever. When the disease was of three days' duration pus began to drain from both ears, and the child complained of severe pain. His mother noticed that he had a squint, and he complained of double vision. Palsy of the external rectus muscle was present on the left side. On January 10 (seven days later), a bilateral simple mastoidectomy was done at the Municipal Hospital of Camden, N. J., by Dr. Klein. The patient remained at the hospital for one week following the operation and was then sent home in apparently good condition. The night of his return home, pain again developed in the right eye, which was very severe and required rather strong sedatives for relief. The right ear began to drain profusely. The pain became a generalized headache, which was more prominent over the left frontal region. The left eye hurt rather severely during these attacks. The pain was nocturnal, deep-seated, retro-orbital and persistent, and the child was referred to this hospital for study. A diagnosis of Gradenigo's syndrome on the left side and possible petrositis was made on admission.

Examinations.—On physical examination the temperature was 100 F.; the pulse rate, 110, and the respiratory rate, 20. The child was lying comfortably in bed with a mastoid bandage over both ears. He was rather intelligent and was able to give an accurate account of his illness. He was attentive and well oriented. The skin was warm and dry. The whole body was desquamating. The sclerae were clear. The pupils were equal and regular and reacted promptly to light and in accommodation. There was no ptosis or gross defects of the visual fields. There was palsy of the left external rectus muscle. Other physical findings will be detailed.

Examination by Dr. Ersner showed that bilateral mastoidectomy had been done seventeen days previously, following an attack of scarlet fever. The patient had weakness of the sixth nerve prior to the operation on that side. Diplopia was also present. These signs and symptoms subsided and recurred again one week following operation. The mastoid was of the large cell type and coalescent. At the time of examination the patient had profuse suppuration from the right middle ear and the right mastoid wound. There was also some drainage from the postauricular wound of the left side. There were palsy of the sixth nerve, diplopia on the left and supra-orbital pain and pain in the face and teeth on the left.

Fig. 8 (case 8).—Note the evidence of inflammatory changes in the petrous tip of the right temporal bone.

Neurologic examination (Dr. M. Moore) revealed the following pertinent data: (1) normal cranial nerve function except for (2) palsy of the external rectus muscle and the disturbance of the fifth nerve as described, (3) normal reflexes throughout, and (4) normal sensory and cerebellar findings. According to Dr. Moore, the patient presented a Gradenigo's syndrome on the left, secondary to a possible petrositis, which at the time seemed to be resolving satisfactorily. The comparatively moderate weakness of the left external rectus muscle and the cessation of the facial pain in association with the clinical improvement, as manifested by the temperature and the pulse rate, indicated a resolution of the involvement of the petron. Conservative treatment and watchful waiting were indicated at this time.

Ocular examination (Dr. Lillie) showed the reflexes to be normal. There was limitation of the movement of the left eye in the field of the external rectus muscle, with diplopia increasing in that field. The perimetric fields were normal. The diagnosis was Gradenigo's syndrome on the left side.

Roentgenographic examination, by Dr. A. K. Merchant, which was interesting and especially important, revealed many mastoid cells remaining on both sides. All of the cells were abnormally dense, the density being a little more pronounced in the remaining cells of the left mastoid than in those of the right. There was nothing to indicate definitely whether or not active suppuration remained on either side at this time.

The appearance of the tip of the petrous portion of each temporal bone suggested that there were small cells on both sides. The presence of inflammatory changes in the tip of the petrous portion of the right temporal bone was suspected, but the findings were not absolutely conclusive (fig. 8). The jugular foramina were within normal limits, the right side being slightly larger than the left. The same was true of the lateral sinuses.

It was concluded that there was roentgenographic evidence of persistent exudative mastoiditis in the remaining cells of both mastoids and questionable but possible petrositis on the right side.

Progress.—After the completion of this study, it was decided to observe the patient carefully. He remained in this hospital for ten days, during which time there was no evidence of any orbital pain or pain in the face. He was able to sleep well. The mastoid wounds were redressed daily and gradually became dry. The temperature remained low, never rising above 100 F. (rectal). His condition being good, the patient was discharged from the hospital on March 11, ten days after admission. At the time of his discharge, the palsy of the left sixth nerve was still present, but there was absence of diplopia, of ocular pain and of pain in the face or in the teeth. Both mastoid wounds and middle ears were dry. The patient was referred to Dr. Klein for follow-up study and redressing.

He remained well until one week following his discharge, at which time there was a recurrence of symptoms. At this time, hesitating to delay any longer, Dr. Klein reoperated on both mastoids. At the second operation, he found that the mastoid wounds were free from pus. Both cavities were full of firm granulation tissue. This was found obstructing the antrum, which he curetted. A few remaining cells were present in the zygomatic regions, which he removed. Following this, the child went on to an uneventful recovery.

Summary.—The following points summarize this case: (1) scarlet fever followed by bilateral suppurative otitis media; (2) onset of palsy of the left external rectus muscle and bilateral mastoiditis; (3) bilateral simple mastoidectomy; (4) relief from symptoms and cessation of discharge; exacerbation one week after operation, with palsy of the left sixth nerve, orbital pain, pain in the face and cheeks; (5) roentgenographic evidence of changes in the petrous portion of the right side, and a persistent exudative mastoiditis of both sides, normal lateral sinuses and jugular foramina, the right being slightly larger than the left; (6) profuse suppuration from both mastoid wounds and middle ears; (7) Gradenigo's syndrome on the left and roentgenographic evidence of right-sided petrositis, (8) expectant treatment, with relief from pain, diplopia and the bilateral suppuration, and (9) exacerbation of symptoms one week after the patient was sent home from the hospital; reoperation on both mastoids and uneventful recovery.

Comment.—To our minds, this case is especially important, not from the point of view of Gradenigo's syndrome, which is fairly common,

but owing to the fact that the syndrome occurred on the left side and the roentgenogram revealed changes in the apex of the petrous pyramid on the right. The patient presented no symptoms except aural discharge on the right, whereas on the left the various findings as described were present. The cause of Gradenigo's syndrome has often been ascribed to a lesion in the petron. Here in one patient Gradenigo's syndrome occurred on the left without petrositis and petrositis on the right without Gradenigo's syndrome. To our knowledge a case of a similar nature has not been reported in the literature, i. e., one that presented two distinct entities, corroborated by roentgenographic and clinical findings. This should help to substantiate the belief of other workers, such as Kopetzky, Uffenorde and Vogel. Uffenorde [17] stated, in 1929, repeating the opinion he expressed in 1920, that "while Gradenigo's triad is seldom found in petrosal tip suppuration, trigeminal irritation is very often evident," and he further stated that "we should depend much less on the abducens palsy as evidence of peri-labyrinthine suppurative foci." Vogel [17] stated that "where a symptom complex is beclouded by other factors or varies, it is no longer a symptom complex characteristic of a certain condition. Otogenic paralysis of the abducens is not diagnostic of affections of the pyramidal tip, and in itself does not furnish indications for extensive operative procedures on the pyramidal tip."

The following case is another of especial interest for the following reasons: The patient presented definite symptoms of labyrinthitis, as well as roentgenographic evidence of destruction in the mesial end of the petrous apex of the right side.

Case 9.—*History.*—D. C., aged 21, a man, white, was well until one month previous to his admission to the hospital (March 18, 1934), at which time he had an attack of acute follicular tonsillitis and influenza. This was followed by pain in the right ear. On examination of the ear drum a rather red membrane, on the surface of which were several blebs, was seen. These blebs were carefully punctured. The temperature continued to be elevated, and the patient suffered considerably with pain in the right eye and rather severe frontal headaches. At this time evidence of an acute suppurative otitis media was present. Myringotomy was done on the right side under general anesthesia. The day following the incision of the drum, the patient complained of nausea. He had several chills; the temperature was 101 F. There was a mixed rotatory nystagmus to the left. At this time a diagnosis of acute labyrinthitis was made, and the patient was referred to the otologic service of Temple University Hospital on March 30, 1934, twelve days after the onset of the illness.

On admission the temperature was 101.2 F.; the pulse rate, 93, and the respiratory rate, 20.

Examinations.—Physical examination revealed a man of 20, suffering rather acute pain in the right ear and mastoid regions. He was unable to sit up owing to the nausea and vertigo that followed. The general physical examination of the

17. Quoted by Kopetzky and Almour.[7]

pulmonic and cardiovascular system gave negative results. The eyes presented a mixed rotatory nystagmus to the left. The pupils were equal and reacted to light and in accommodation. The extra-ocular movements were full in all directions. There were a positive Kernig sign, a suggestive Brudzinski sign and a slight nuchal rigidity.

A spinal puncture at this time showed the fluid to be under increased tension, with a cell count of 75 white blood cells, of which 62 per cent were polymorphonuclear leukocytes. The sugar was 75 mg. per hundred cubic centimeters; the chlorides, 700 mg. No organisms were present on direct smear or in culture.

Fig. 9 (case 9).—Note the evidence of a marked inflammatory process in the mesial end of the right petrous bone.

Roentgenographic examination by Dr. W. E. Chamberlain, made on April 30, was reported as follows: The appearance of the left mastoid was entirely normal. The right mastoid exhibited a hazy clouding, which is characteristic of exudative mastoiditis, but the mesial end of the right petrous bone, which was quite thoroughly pneumatized, exhibited marked disease. It was possible to say that the inflammatory process was more intense in the mesial end of the petrous bone than in any other part of the mastoid structure, and the presence of actual bone destruction at this point was strongly suspected (fig. 9). Both jugular foramina were rather smaller than normal, with the right slightly larger than the left.

In view of the foregoing findings in the roentgenogram, plus the beginning meningeal involvement and the labyrinthine symptoms, it was decided to perform a mastoidectomy without delay.

Operation.—On March 31 (thirteen days after the onset of the illness and the day following the admission to the hospital), an extensive simple mastoidectomy was done. The mastoid process was found well pneumatized, cells extending into the zygomatic region, anteriorly and posteriorly behind the lateral sinus into the occipital region and peribulbar and perilabyrinthine cells. There were considerable pus and necrosis of cellular septums, which were thoroughly exenterated. The lateral sinus was exposed from the sigmoid to the jugular bulb and was found to be normal. The dura of the middle fossa was exposed, and it seemed normal. The incus was removed in order to take the tension off the oval window and to avoid the necessity of performing a radical mastoidectomy. A spinal puncture was done while the patient was under the anesthetic, and the fluid preserved for study.

Course.—The patient's immediate postoperative condition was good. The report on the spinal fluid, made at the time of the operation, showed a reduction in the number of cells. The chemistry was entirely normal. The day following the operation the patient had reacted well, although the temperature rose to 104 F. On April 1, the nuchal rigidity had practically disappeared, the headaches were gone, and there was only a slight residual nystagmus present. On April 4, the fourth postoperative day, the temperature was practically normal, the nystagmus had disappeared, and the patient felt better. On this day he was given a blood transfusion of 300 cc. From this time on, convalescence was uneventful, and the patient was discharged on March 18, nineteen days after admission to the hospital and thirty days after the onset of the illness. Follow-up study made up to the present time has shown no recurrence of the aural pathologic process.

Summary.—The following outline summarizes the case: (1) acute tonsillitis and influenza; (2) influenzal otitis, with blebs; (3) acute suppurative otitis media, myringotomy and profuse bleeding; (4) onset of labyrinthine symptoms; nausea, vomiting, vertigo and mixed rotatory nystagmus to the left; (5) meningeal signs, spinal puncture and increased cell count of the spinal fluid (two punctures); (6) an extensively pneumatized mastoid, with cells extending into the petrous apex, and roentgenographic evidence of bone destruction in this region; (7) extensive simple mastoidectomy, with recovery, and (8) removal of the incus.

Comment.—The patient presented an extensively pneumatized temporal bone, with cells that reached in every direction. It is easily understood, therefore, how infection could spread via the perilabyrinthine route into the apex of the petrous portion of the temporal bone. It is interesting to note that in the roentgenogram there was more evidence of destruction in the petrous portion of the temporal bone than in the mastoid process. It is the presence of the labyrinthine symptoms that makes us believe that the avenue of infection was through the perilabyrinthine route. It might also be stated that in this instance we hold with Kopetzky and Almour that the labyrinthitis is not a true labyrinthitis, but due to a perilabyrinthitis.

The type of mastoiditis that the patient had was a thrombotic, hemorrhagic type, and it is possible that the small size of the lateral sinuses

Data on Cases Reported

	Case	Age	Sex	Side	Ocular Pain	Optic Sepsis	Aural Discharge	Quiescent Postoperative Period	Terminal	Roentgenographic Involvement of Petron	Operation			Gradenigo's Syndrome	Meningeal Symptoms	Labyrinthine Symptoms	Size of Jugular Foramina and Lateral Sinuses			Eyeground	Diplopia	Profuse Bleeding After Myringotomy
											Simple Mastoidectomy	Myringotomy	Kopetzky-Almour				Right	Left	Equal			
1	McG.	20	F	R	+	+	+	+	Recovery	R +	+	+	—	—	—	—	.....	Larger	.....	Normal	—	—
2	W. S.	21	M	R	—	+	—	—	Recovery	R +	+	—	—	—	—	—	Slightly larger	.....	About equal	Normal	—	—
3	M. W.	20	F	R	—	—	—	—	Recovery	R +	+	+	—	—	—	—	Slightly larger	.....	About equal	Normal	—	—
4	R. K.	39	M	R	+	+	+	+	Recovery	R +	+	+	+	+	—	—	?	?	?	Slightfulness of retinal vessels	+	—
5	M. L.	25	M	L	+	+	+	—	Recovery	L +	+	+	—	—	—	—	Larger	.....	.....	Not examined	—	+
6	H. B.	8	M	L	+	+	+	+	Recovery	L +	+	+	—	—	—	Slight vertigo and dizziness	Larger	.....	.....	Normal	—	—
7	H. W.	6	F	R	—	+	+	—	Recovery	R +	—	+	—	—	—	—	Larger	.....	.....	Normal	—	+
8	C. N.	8	M	R	+	+	+	+	Recovery	R +	Bilateral +	+	—	L +	—	+	Larger	.....	.....	Normal	+	—
9	D. C.	21	M	R	+	+	+	+	Recovery	R +	+	—	..	—	+	+	Smaller than normal right	.....	.....	Normal	—	—
	Total.....				7	6	8	7	5		9	8	7	1	2	1	2	7			2	2

and jugular foramina, and especially the involvement of the right side, which was the larger, produced an increase of congestive circulatory symptoms.

Our thought in the removal of the incus was to release tension on the stapes and thus on the oval window, and in this manner to try to relieve the labyrinthine symptoms and to avoid a radical mastoidectomy.

SUMMARY AND CONCLUSIONS

1. A review of the literature relative to the involvement of the apex of the petrous portion of the temporal bone has been given.

2. The relationship of Gradenigo's syndrome to petrositis has been emphasized: (*a*) Gradenigo's syndrome is independent of petrositis. (*b*) Petrositis may produce the Gradenigo's syndrome. (*c*) Petrositis may exist without any symptoms.

3. It has been stated that Gradenigo's syndrome, or petrositis, may be due to venous congestion of the temporal bone resulting from venous circulatory disturbances.

4. In many instances the roentgenologist is unable to distinguish between petrositis, exudative petrositis and congestion of the petrous portion of the temporal bone due to venous anomalies of the skull.

5. According to Wittmaack, Profant, Druss, Kopetzky and others, true petrositis can occur only in a pneumatized temporal bone.

6. In a diploic temporal bone, the lesion is usually osteomyelitic rather than coalescent.

7. Petrositis, as revealed on roentgenographic examination, does not necessarily indicate surgical intervention.

8. The Kopetzky-Almour operation or other operations on the petrous portion of the temporal bone are not our first consideration, as many patients recover after simple mastoidectomy and myringotomy.

9. Key films are essential for the following reasons: (*a*) to determine the type of temporal bone, whether pneumatic, sclerotic or diploic; (*b*) to determine the size of the lateral sinus and jugular foramina, and (*c*) for the roentgenographic visualization of the temporal bone for a permanent record. Should signs and symptoms appear, the plates become of intrinsic value.

10. A series of nine cases were presented, each of which showed definite roentgenographic evidence of involvement of the apex of the petrous portion of the temporal bone. Recovery occurred in each of these cases. (*a*) One patient (R. K.) recovered following a Kopetzky-Almour operation. (*b*) Another patient recovered as the result of several myringotomies. (*c*) Rare judgment and care are necessary in deciding on the treatment in each case. Radical surgical procedure should always be based on definite indications.

ABSTRACT OF DISCUSSION

DR. JOHN W. CARMACK, Indianapolis: My co-workers and I have dissected the petrous tips in forty-seven adult cadavers, or ninety-four tips in all. There were pneumatic cells in the pyramid in 32 per cent. Here a pneumatized tip implies the extension of air cells, medial to the labyrinthine capsule.

It is important from the standpoint of pathologic changes, symptoms and treatment that all pyramids in which air cells extend past the labyrinthine capsule be classified as pneumatized. In all forty-seven cadavers the pneumatization was bilateral and occurred without exception with well pneumatized mastoid processes. Pneumatic extension occurred about equally from in front and behind the labyrinthine capsule.

There is every anatomic reason for extension of an inflammatory process into the cells of the pyramid, with otitis media, similar to that occurring in a mastoid process.

To substantiate further the fact that pathologic changes occur in the cells of the petrous tip, with simple otitis media and without symptoms of petrositis, I wish to show a photomicrograph of a cell of the petrous apex of a patient who died of pneumonia five days after incision of the ear drum for suppurative otitis media. There were no symptoms of petrositis or mastoiditis, but at postmortem examination there was swelling in the mucous membranes of the mastoid and the cells of the petrous tip, without evidence of pus in the cells. The mucosal swelling and exudate into the cells would certainly show roentgenographic changes.

Microscopic study of this case indicates, first, an inflammatory reaction in the mucosa of the cells of the petrous tip rather than venostasis; second, extension of the inflammation into the cells of the petrous tip by way of the pneumatic spaces.

Reasoning by analogy, one might expect to find some roentgenographic changes in the petrous tip of any number up to approximately one third of all suppurations of the middle ear in adults. From increasing pathologic roentgenographic and clinical studies, it is becoming more evident that the roentgenogram alone is not a safe guide to surgical exploration in the petrous apex.

I admit being skeptical of the roentgenographic report in one or two of the authors' cases in which the formation of pus was reported present in the tip, and in which recovery promptly followed operation on the mastoid.

Regarding the mechanism of the production of symptoms in disease of the petrous tip, such as palsy of the sixth nerve and pain in the trigeminal nerve, I am of the opinion that it is the result of pachymeningitis, identical with the periostitis, occurring over the mastoid process in mastoiditis.

If congestion of the veins due to lack of return of the venous blood by way of the lateral sinus and jugular veins were important, why does one not see palsies of the sixth nerve after thrombosis of the sigmoid or after ligation of the jugular vein? It is true that paralysis of the sixth nerve does not mean petrositis, but its presence with the characteristic pain in the trigeminal nerve and other clinical evidence of petrositis indicates localized meningitis.

The value of this paper is that it calls attention, first, to the fact that roentgenographic evidence alone is never a justification for operation on the petrous tip. Second, that a thorough operation on the mastoid may prevent extension of the infection of the petrous tip or even permit a cure in the early stages of disease of the petrous pyramid. I commend a conservative attitude in any questionable case.

On the other hand, I voice an emphatic warning against delayed drainage of the pyramid by some method after definite clinical evidence of suppuration is present, as meningitis will occur in a high percentage of cases in which operation is not performed.

I am of the opinion that in many cases of suppuration of the petrous tip satisfactory drainage can be obtained by one of the less radical procedures described, but that all methods described to date have a definite place, depending on the anatomic conditions presented.

Dr. B. H. Nichols, Cleveland: For several years it has been the practice of my co-workers and me to include an examination of the sphenoid bone in all cases in which an examination of the nasal accessory sinuses is made. In reviewing these cases we have had ample opportunity to study and compare a large number of temporal bones and to observe the normal roentgenographic variations in the petrous portion. Such a survey has shown marked variation in the anatomy of this bone, particularly as to the presence of pneumatized cells and their extent, which varied from a few cells in the tip to marked pneumatization, or in many instances to the absence of cells in one or both petrous bones. However, such a survey shows a rather consistent similarity in the two sides. The normal variations are of sufficient frequency to make one extremely cautious in rendering an opinion from the roentgenographic examination alone, and we feel that the roentgenogram should be considered only as an aid in the diagnosis of petrositis.

It has been our practice for the past two years to study the petrous bone at the first examination in all cases of suspected mastoiditis, as recommended by the authors of this paper. We found this helpful in some cases, but in most cases of well marked mastoiditis the cells of the tip are occluded. We are unable to determine whether this is an actual infection with edema, perhaps with some serum in the cells, or an unpneumatized tip. Hence we feel that the sphere of roentgenologic diagnosis of petrositis is quite limited and that operation should be considered only when there are definite symptoms. Reexamination after mastoidectomy shows that most of the roentgenographic signs have disappeared, as compared with the results of the initial examination. When such is not the case, the reexamination is of great help, and from such an examination definite aid may be obtained.

The authors of this paper have clearly stated the various changes in the normal bone encountered and have interpreted their importance correctly.

Dr. Henry K. Taylor, New York: The roentgenographic examination is an aid, and while it may yield considerable useful information, the final diagnosis does not rest on it. The term "petrositis," in my opinion, is not a good one. It includes all inflammatory conditions of the petrous bone. The subject under discussion should be called either empyema or a suppurative lesion in the petrous apex. I believe that either one of these terms describes the complication of an otitic infection in which we are all particularly interested. This lesion develops only in a pneumatic bone. An infection in a diploic bone gives rise to osteomyelitis.

Any otitic infection will show, on the roentgenogram, a change in the aeration of a pneumatic petrous pyramid—an increase in density. This change in aeration does not mean that a suppurative lesion exists.

When there is an extension of the infection into the petrous apex, there are definite roentgenographic changes. Drs. Kopetzky and Almour described a familiar symptom complex: a history of mastoidectomy on a well pneumatized bone, normal convalescence with a variable period of well-being, reappearance of the aural discharge, low grade sepsis, involvement of the fifth nerve and pain referred to as behind or in the eye. Roentgenographic changes in the petrous apex

of a destructive nature, associated with the aforementioned symptom complex, indicate a suppurative lesion in the petrous apex, with inadequate drainage. This is the complication which requires surgical intervention to induce adequate drainage, to avoid an intracranial complication. There are similar roentgenographic changes in patients in whom drainage is adequate. The only symptom is a persistent otorrhea. This patient is in no immediate danger and requires no operation to avoid an intracranial complication. This lesion may terminate in chronic otorrhea. A number of cases of protracted otorrhea are due to a suppurative lesion in the petrous apex. Here, the roentgenographic findings are those of a productive nature.

I will conclude by paraphrasing an old saying of Jackson's—"everything that wheezes is not asthma"—as follows: Every change in the roentgenographic appearance of the petrous apex does not indicate a suppurative lesion in the petrous apex.

Dr. M. C. Myerson, New York: If one could answer the question, "When is petrositis petrositis, or when are we dealing with a surgical condition?" one would have accomplished something. I believe that we are passing through a formative period in the evolution of the management of this condition, just as we did in mastoiditis, and that there are bound to be disagreements until enough fundamental work is done to enable one to handle these cases properly.

My co-workers and I were fortunate to have the opportunity to study two hundred petrous bones in one hundred consecutive skulls in fresh cadavers at the Kings County Hospital.

Dr. Carmack asked me whether we had children in this series. I am glad he did, as I believe that our percentage of pneumatization is lower because we had thirty-eight children in our series. However, I should like to show a comparative table of pneumatization of petrous bones, both our findings and those from the literature.

Zuckerkandl studied two hundred and fifty mastoids with no petrous apexes, and Belinoff and Balan studied forty, with a percentage that was previously given us by the essayist. I do not see that one can compare the pneumatization of the mastoid with the pneumatization of the petrous apex in the series of Belinoff and Balan. If Zuckerkandl had studied the mastoid and petrous apex in each of his two hundred and fifty bones, he would have given one something worth while.

This is a diagram [slide] that gives one a fair understanding of the arrangement of the cells in the petrous pyramid, as these cells continue inwardly from the middle ear cavity. There is an arbitrary line *A-B* drawn through the horizontal portion of the facial nerve which divides these groupings into supracochlear and infracochlear cells. The supracochlear are divided into (1) the anterior surface cells, (2) the superior border cells and (3) the posterior surface cells. The infracochlear cells are divided into those anterior to the cochlea, or the tubal cells mentioned by Kopetzky, and the subcochlear cells and those posterior to the cochlea.

We used various dyes, such as methylene blue, green or red ink. In twelve of the bones the dye ran out of the no. 1 series, or the anterior surface group of cells; in fourteen, we found that the subcochlear and the infracochlear cells carried the dye into the mastoid. It is just possible that when those cell groups that I just mentioned, that is, those in the subcochlear and the infracochlear regions, are involved, the infected cells may be able to drain into the mastoid area and, therefore, perhaps a simple mastoidectomy will suffice. However, all who have had the clinical experience of encountering a fistula at the time of operation have not seen a spurt of pus enter the middle ear cavity, but rather have encountered a

softened area and then a spurt of pus. In other words, if there is a petrous pyramid requiring operation, there must be trapped pus in that petrous pyramid, and particulary in the area of the petrous apex.

This slide shows the depth of one of the many bones removed. In these two hundred petrous bones, the average distance from the semicircular canal to the tip of the apex was 3.2 cm. These landmarks are of great value in the roentgenograms to tell where the shadows are. There are elevations and depressions which should be taken advantage of in reading zones and in the surgical approach by the middle fossa route.

There is quite a distance to go before the infected exudate is going to come through.

Dr. Matthew S. Ersner, Philadelphia: A question was asked why palsy of the sixth nerve does not occur more frequently in sinus thrombosis. I could answer this query with an illustration: When one ligates the carotid fractionally, the chances for any intracranial symptoms and paralysis are remote because the contralateral circulation adapts itself gradually. It is only when one ligates the carotid completely at one sitting that the patient is more apt to show changes in the brain with a subsequent paralysis.

Thrombus formation in the lateral sinus is a relatively slow process and during this period of formation the contralateral circulation adapts itself so that it can take care of the circulatory embarrassment. When the catastrophe occurs suddenly, palsy of the sixth nerve is apt to occur, especially where the contralateral venous structures are small.

In another series, we had two cases in which palsy of the sixth nerve developed.

Dr. Taylor mentions mucous membrane in the petrous portion of the temporal bone. In the diploic bone mucous membrane is absent. However, according to Wittmaack and others, wherever mucous membrane is present there is pneumatization.

CYTOLOGY OF NASAL POLYPI

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For many years the question of the etiology of nasal polypi has received the attention of rhinologists. In a recent publication Kern and Schenck [1] gave an excellent survey of the literature and enumerated the theories that have been advanced on the etiology of the so-called mucous polyp. Until recently, chronic nasal infection and sinus suppuration were thought to be the most common etiologic factors, although theories were advanced that polypi were of vascular origin, were due to lymph-vascular disease or were hereditary. More recently, the importance of nasal allergy in the etiology of nasal polypi has been emphasized, and Kern and Schenck found that "mucous polyps are extremely common in allergic conditions of the upper respiratory tract, and that in patients presenting symptoms of non-allergic disease of the upper respiratory tract mucous polyps are comparatively rare, even in the presence of extensive sinus suppuration." Hansel,[2] in a comprehensive study of the histopathology of the nose and accessory sinuses in allergy, pointed out the presence of eosinophils in allergic tissues, and urged that allergic sinus disease, with or without secondary infection, should be differentiated from primary sinus infection with edema, hyperplasia and polypi. Such differentiation is of the utmost importance if efficient treatment is to be adopted, because, although surgical intervention is usually essential in the treatment of chronic infections of the sinuses, it is generally agreed by the more conservative observers that nasal allergy is not of itself a condition requiring surgical intervention.[3]

From the Department of Surgery, Division of Otolaryngology, the University of Chicago.

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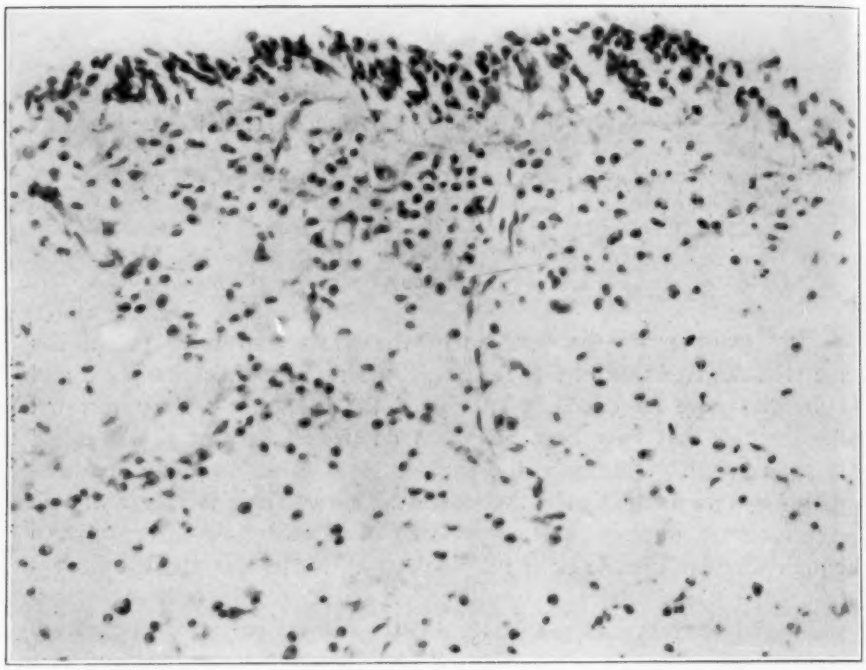

Fig. 1.—Photomicrograph of nasal polypus of type A, from a patient with many allergic sensitivities. There was no clinical evidence of infection. All the polymorphonuclear cells are eosinophils. There are a few plasma cells. The basement membrane is thickened; × 250.

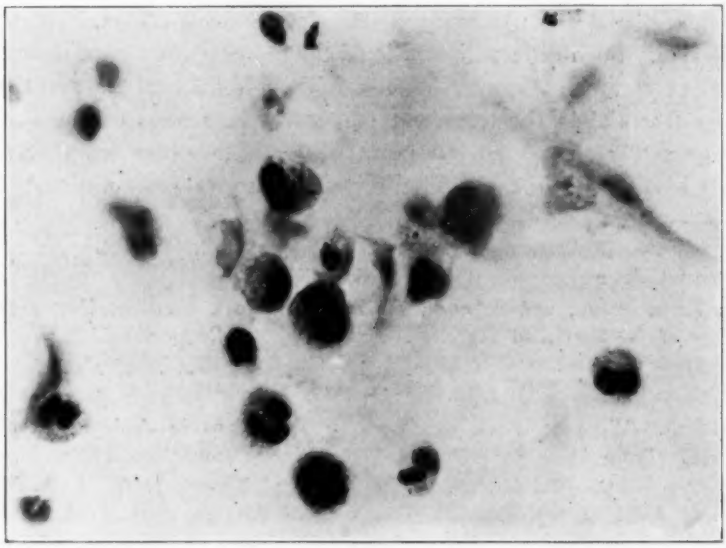

Fig. 2.—From the same section as figure 1; × 1,200.

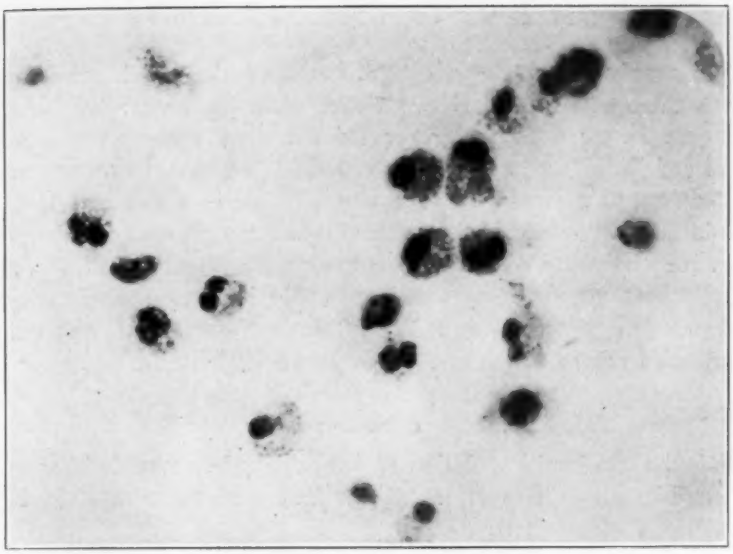

Fig. 3.—From a case similar to that shown in figures 1 and 2; × 1,200.

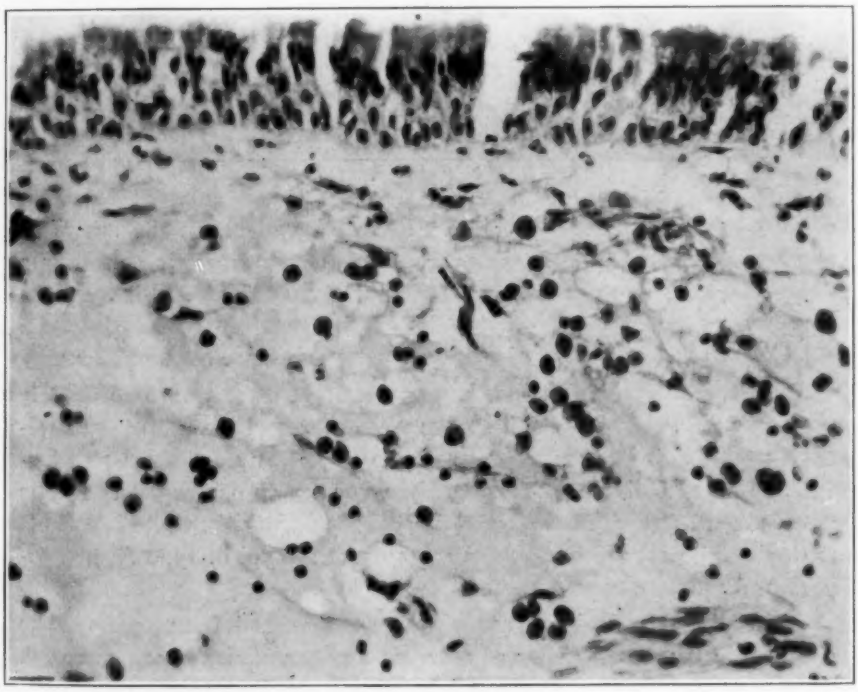

Fig. 4.—Photomicrograph of a nasal polypus of type A, in which there was infection in the sinuses. Eosinophils and neutrophils are present in approximately equal numbers. The basement membrane is thickened; × 350.

The results of cytologic examinations of the nasal secretions in various cases of rhinitis [4] showed: that eosinophils were the predominant cells in cases of vasomotor rhinitis, that neutrophils were the typical cells in infection and that the ratio of eosinophils to neutrophils was lowered in the cases of vasomotor rhinitis in which infection was superadded. It was decided to extend this cytologic study to the polypi which are admittedly the result of chronic rhinitis and sinusitis, in order to determine the types of cells present in polypi and to ascertain whether, by correlation of the clinical and cytologic observations, any definite factors with regard to the etiology might be established.

CYTOLOGY

Polypi were fixed in solution of formaldehyde, embedded in paraffin and cut in sections 10 microns thick. The sections were stained with

TABLE 1.—*Clinical Observations on Seventy-Five Cases with Nasal Polypi*

	Type A (44)		Type B (31)	
Pus in nose		15		31
No pus in nose		29		0
Unilateral distribution		0		14
Bilateral distribution		44		17
Allergy: complete investigation		26		3
Reaction to skin tests positive	15		0	
Reaction to skin tests negative	11		3	
Blood: eosinophilia increased		11		1

Maximow's hematoxylin-eosin-azure stain. The epithelium varied from ciliated columnar to low cuboidal. Immediately beneath the basement membrane was the greatest number of cells; in the deeper part of the polypi the cells were frequently, but sparsely, scattered. Plasma cells were numerous in all the polypi; the other cells seen were eosinophils, polymorphonuclear neutrophils, lymphocytes and large mononuclears.

In examining polypi it became apparent that there were two distinct types: A, in which eosinophils were numerous and often the predominating cells, and B, in which there were few if any eosinophils and in which the predominating cells were lymphocytes or neutrophils. It was noted that in type A the basement membrane was thick, while in type B it was usually not thickened. In some of the polypi of type A, neutrophils were almost as numerous as eosinophils.

After the histologic report of the sections had been written, the clinical history of the case was obtained and the clinical findings noted. Table 1 shows the correlation of the clinical findings in the two types.

4. Lindsay, J. R., and Walsh, T. E.: Nasal Secretions: The Value of Cytologic Examination to the Rhinologist, Arch. Otolaryng. **17**:783 (June) 1933.

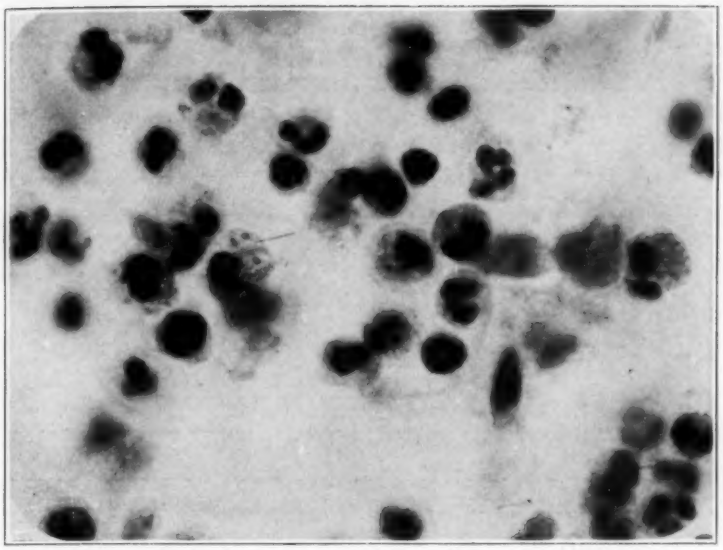

Fig. 5.—From the same section as figure 4; × 1,200.

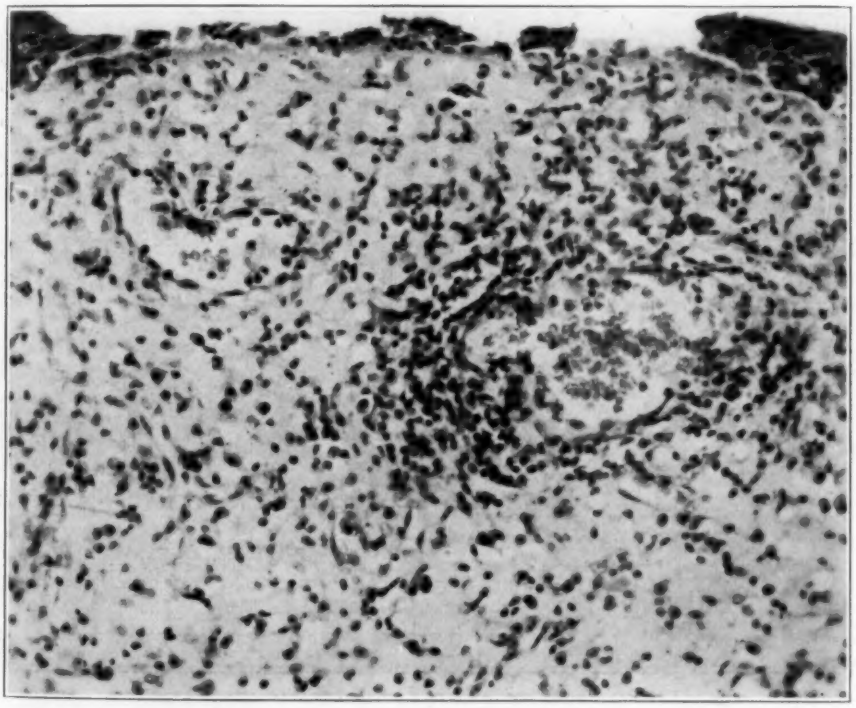

Fig. 6.—Photomicrograph of a polypus of type B. The patient underwent a complete examination for allergy. No positive reactions were found. Both antrums were full of pus and polypi. The cells are neutrophils and lymphocytes. Occasional eosinophils are present. The basement membrane is not thickened; × 250.

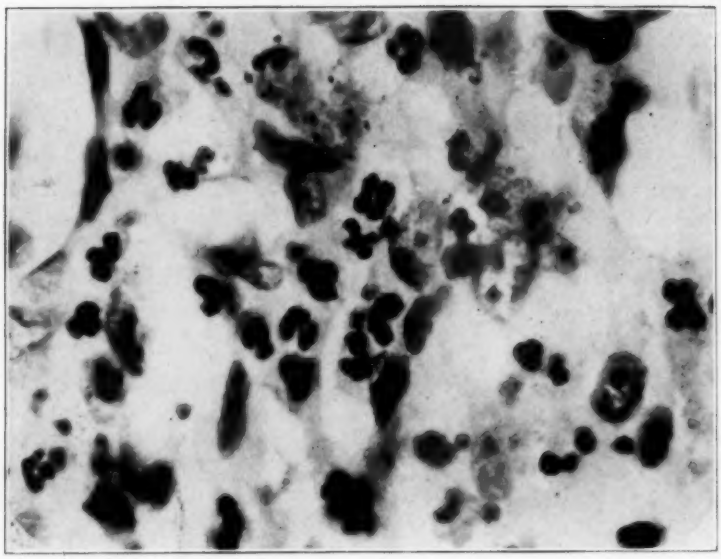

Fig. 7.—From the same section as figure 6; × 1,200.

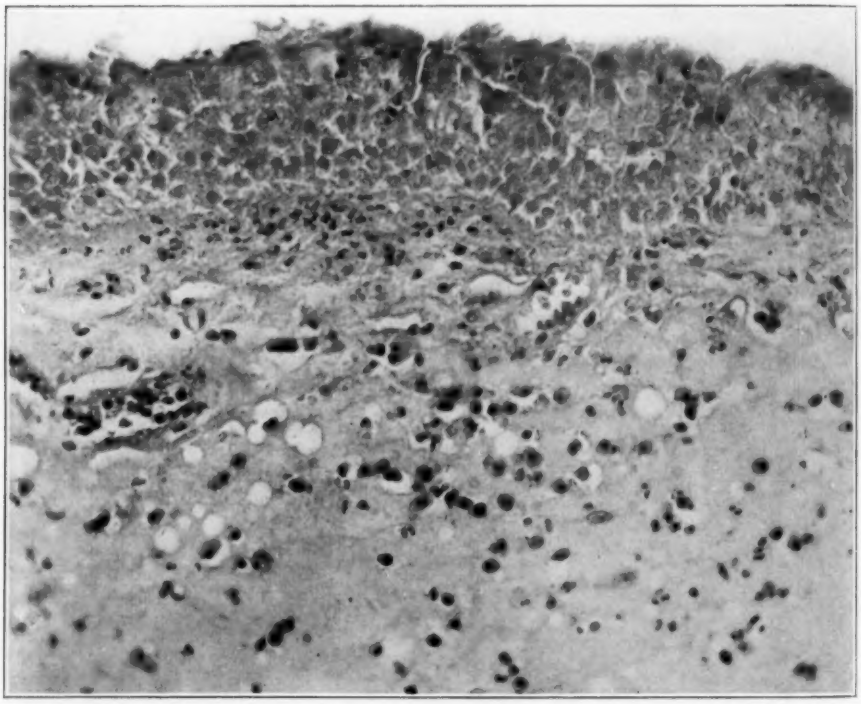

Fig. 8.—Photomicrograph of a polypus of type B from a case of chronic sinusitis of the right antrum. Note the hyperplasia of the epithelium and the fact that there is no thickening of the basement membrane. All the invading cells are neutrophils. The pathologic condition was unilateral.

Pus was found in the nose or sinuses of every patient who had polypi of type B. In fourteen cases the pathologic process was confined to one side only; in seventeen the condition was bilateral. In only three cases was a complete allergic examination made, and no positive reaction to the tests was found. One patient had increased blood eosinophilia (15 per cent).

There were fifteen cases of polypi of type A in which pus was seen in the nose or sinuses, and twenty-nine in which the clinical diagnosis was vasomotor rhinitis and there was no evidence of infection. In all forty-four cases in type A the pathologic condition was equally evident on both sides.

Complete allergic examination was carried out in only twenty-six of the forty-four cases in the type A group, and of these cases positive skin reactions were found in fifteen and no positive reactions in eleven. Of the eleven patients who failed to show a positive skin reaction, four had asthma, one gave a definite story of autumn hay fever, four had a markedly low blood pressure and basal metabolic rate and two, apart from the nasal condition, showed no abnormality. In eleven cases in type A the blood eosinophilia was above 5 per cent.

TABLE 2.—*Results of Surgical Treatment in Patients with Nasal Polypi*

Condition Following Surgical Treatment	Type A	Type B
Cure to marked improvement	13 (35%)	20 (77%)
Fair improvement	7 (19%)	2 (8%)
No improvement	17 (46%)	4 (15%)

Surgical treatment, ranging from simple removal of the polypi to radical antral and ethmoid operations, was carried out in thirty-seven patients with type A polypi and in twenty-six with type B polypi. The results of such surgical treatment are shown in table 2.

COMMENT

The occurrence of an eosinophilic infiltration into the cutaneous wheals formed by the injection of the appropriate allergen in allergic persons was demonstrated by Kline and his co-workers.[5] They also showed that similar eosinophilic infiltration occurred in sensitive persons following the injection of histamine, and that in nonallergic persons no local eosinophilia was obtained. Knott and Bruce Pearson [6] confirmed

5. Kline, B. S.; Cohen, M. B., and Rudolph, J. A.: Histologic Changes in Allergic and Non-Allergic Wheals, J. Allergy **3**:531, 1932.

6. Knott, F. A., and Bruce Pearson, R. S. B.: Eosinophilia in Allergic Conditions, Guy's Hosp. Rep. **84**:230, 1934.

their observations and suggested that the concentration of eosinophils in the fluid of wheals following the injection of histamine be used as a diagnostic test for allergy. The presence of eosinophils in allergic nasal tissues has been pointed out by Weille,[7] Hansel [2] and others. Murphy [8] described eosinophilia of the nasal mucosa of guinea-pigs as a constant observation in anaphylactic changes in those animals. The accumulated evidence seems to indicate that the presence of eosinophils in tissues is indicative of allergy.

The finding of two types of polypi in this series of seventy-five cases, the one invariably associated with infection, the other more often with no clinical evidence of infection but frequently associated with demonstrable allergy, and, further, the approximately equal number of the two types do not support the view of Kern and Schenck that "in patients presenting the symptoms of non-allergic disease of the upper respiratory tract mucous polyps are comparatively rare even in the presence of extensive sinus suppuration." Nor, on the evidence of our observations, can it be supposed that polypi are more commonly due to infection than to allergy. Hansel's view that polypi may be due either to allergic disease or to infection coincides with our observations.

Eleven cases in this series showed many eosinophils in the polypi, with no positive reaction to the skin tests. That the reactions were negative does not, we believe, preclude the possibility that allergy was present. In one case the patient gave a definite history of autumn hay fever, and yet showed no positive reactions to tests with pollens. In another case, not in this series, skin tests showed no positive reactions on one occasion, though on repetition at a later date many positive reactions to the allergens previously tested were found. There were, in this series, eight patients with polypi of type A, in whom a markedly low basal metabolic rate and a low blood pressure were seen. Four gave definite positive reactions to skin tests and four gave negative reactions to all skin tests. Huber and Harsch [9] reported such cases as so-called nonallergic vasomotor rhinitis. In view of the cytologic findings it seems difficult, as Hansel suggested, to separate them from the allergic group.

There is no evidence that throws light on the origin or function of eosinophils. Scott Williamson,[10] writing of nasal polypi, described an

7. Weille, Francis L.: Asthma: The Pathology of Allergic Tissues as Seen in the Nose and Accessory Sinuses, Arch. Otolaryng. **12**:785 (Dec.) 1930.

8. Murphy, William E.: Anaphylactic Changes in the Nasal Mucosa of Guinea-Pigs, Arch. Otolaryng. **13**:842 (June) 1931.

9. Huber, Harry L., and Harsch, George F.: The So-Called Non-Allergic Vasomotor Rhinitis, J. Allergy **5**:432 (May) 1934.

10. Williamson, G. S.: The Genesis of Nasal Polypi, J. Laryng. & Otol. **47**: 667, 1932.

"eosinophilic adenoma" and said, "the eosinophilia is the local product of the cells of the reticulo-endothelial system." He offered no proof for this statement. If the eosinophils were formed locally from the cells of the reticulo-endothelial system, it seems reasonable to suppose that cells in various stages of maturity would be seen in the polypi. In all the sections we examined, however, only adult eosinophils, such as are seen in circulating blood, were observed. Biggert [11] reviewed the theories advanced on the subject of eosinophilia in the tissue and from his experiments concluded that the eosinophilic cell of tissues is an emigrated eosinophilic leukocyte, and that eosinophilia occurs in local tissue at the expense of the circulating eosinophils. He also found that local eosinophilia may persist after the disappearance of general eosinophilia. This possibly explains the absence of increased eosinophilia of the blood in so many of our cases.

The association of infection with eosinophilia in the tissues would seem to indicate that infection was superimposed on an allergic membrane or that there was present an allergic sensitivity to the infecting organism. In one case the patient showed positive cutaneous reactions to organisms recovered from the antrums. In either event the removal of the infection is to be desired, and in both, the determination of the offending allergen is essential to successful treatment. The results of the surgical treatment of thirty-five patients with polypi of type A indicate how guarded the prognosis should be. On the other hand, the results of similar surgical treatment of twenty-six patients with polypi of type B are distinctly good.

SUMMARY AND CONCLUSIONS

The polypi from seventy-five cases of nasal polyposis were examined cytologically, and it was found that they could be divided into two types: A, those with many eosinophils in the tissue, and B, those in which eosinophils were scarce or absent. Correlation of the clinical and histologic observations in these cases revealed that the polypi in type B were invariably associated with infection in the nose or accessory sinuses, while those in type A were often found in persons free from infection and frequently associated with demonstrable allergy.

Accumulated evidence points to the fact that eosinophilia in the tissue is associated with allergy, and it is suggested that the presence of eosinophils in the nasal polypi is indicative of allergy. In cases in which eosinophilia in the nasal polypi is associated with infection, the infection

11. Biggert, J. G.: Some Observations on the Eosinophilic Cell, J. Path. & Bact. **35**:799, 1932.

is secondary in the allergic membrane or possibly there is an allergic sensitivity to the infecting organism.

Surgical intervention in the two types gives very different results. The results in patients with polypi having few or no eosinophils were distinctly satisfactory, while those in patients with polypi having many eosinophils were mostly disappointing.

We conclude therefore that: 1. The histologic examination of nasal polypi with regard to the presence or absence of eosinophils offers a simple method of determining whether the polypi are due to infection or whether they are the product of nasal allergy.

2. The result of surgical treatment of polypi with few eosinophils is good, but in the presence of many eosinophils is distinctly poor.

PARALYSIS OF THE LARYNX DUE TO LEAD POISONING

INCLUDING A CONTRADICTION OF "SEMON'S LAW"

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Poisoning due to lead has been known since before the pre-Hippocratic era. The custom of using vessels made of lead for purposes of storing and preparing wines was probably the greatest contributing factor in the causation of this disease in early times. The ancient Greeks and Romans not only used lead containers for their wines, but also added a piece of lead for purposes of preservation.

The general subject of lead poisoning was admirably dealt with in the classic work of Tanquerel,[1] published in 1839, and in numerous other monographs and texts of more recent date.

In 1572, there was an epidemic of lead poisoning in the south of France which closely resembled the Devonshire colic of the eighteenth century in that its probable source was the use of lead in the cider and wine presses. In 1616, François Citois described Poitou colic, which, long after was found to be the same as lead colic. In 1739, Huxham described the Devonshire colic, which followed the drinking of cider, without, however, ascertaining its true cause. It was discovered by Sir George Baker, who noticed that the colic at cider-time endemic in Devon was somehow correlated with large pieces of lead used in the vats and cider presses, which were not so employed in other counties of England. Cadwalder wrote an essay, which Benjamin Franklin printed in 1745, on the West Indies dry gripes. This was an account of colic and paralysis from the habitual use of Jamaica rum distilled, as Franklin showed in 1786, through lead pipes. In 1840, Burton called attention to the blue line along the gums in lead poisoning.

Much is known concerning the mechanism by which the neuromuscular apparatus is involved in poisoning by lead, although there is no unanimity of opinion on the subject. The following is extracted from the work of Aub, Fairhall, Minot and Reznikoff.[2] In 1879, Friedlander described a degenerative process in which there was a marked

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From the Department of Otolaryngology, Kings County Hospital.

Read before the American Laryngological Association, Cleveland, June 9, 1934.

1. Tanquerel des Planches, L.: Traités de maladies de plomb, ou saturnines, Paris, Ferra, 1839.

2. Aub, J. C.; Fairhall, L. T.; Minot, A. S., and Reznikoff, Paul: Lead Poisoning, Baltimore, Williams & Wilkins Company, 1925.

fibrous replacement of connective tissue of the muscle tissue. He thought that involvement of the muscles preceded involvement of the nerves. Later, Messing demonstrated by microscopic studies that the lesions in the muscles were more marked than in any other part of the neuromuscular apparatus. He believed that lead caused a specific inflammatory lesion of the muscle. Other investigators have expressed the belief that the metallic poison acts on the walls of the blood vessels so that they rupture readily into the nerve and muscle tissue. Most believe, however, that the primary lesion is in the nervous system. Some believe that the spinal cord is involved, while others think that the involvement occurs in the peripheral nerves. It is agreed by most workers that the lesion is generally complex. Hyslop and Kraus believed that the lesion takes the form of a neuronitis which can occur anywhere in the neuromuscular apparatus.

Experimental work done by Aub, Fairhall, Minot and Reznikoff suggests that fatigued muscles are more prone to lead paralysis. According to them, lead is transferred by the blood as an insoluble phosphate in the colloidal state and is converted into a lactate by lactic acid. The soluble lactate comes in contact with the inorganic phosphates in the muscle, so that lead is precipitated as an insoluble phosphate. This reaction depends on the relative amounts of lactate and phosphate present in the muscle. It suggests that susceptibility to lead palsy depends on chemical reactions between lead and the metabolic products of muscular activity. Edinger observed the occurrence of palsy in the muscles most used. It has been demonstrated, however, that distinct lesions occur in the peripheral nerves.

It is questionable whether the premise of these workers will obtain in the cases of laryngeal paralysis due to lead poisoning. If the theory of involvement based on muscular activity is true, then the adductor muscles of the larynx should be most frequently involved, because they are in much more frequent and more active use than the abductors. Speech and the movements which require fixation of the glottis to hold temporarily the intrathoracic pressure, require action of the adductors, while the abductors are used only slightly in quiet respiration and more during forced inspiration.

No tenable explanation has been proposed for involvement of the laryngeal structures. On a basis of what is known of lead paralysis generally the laryngeal lesions fall into four groups: neuropathic, myopathic, encephalopathic and a combination of the neuropathic and myopathic.

Although paralysis of the muscles of the larynx is rare, Tanquerel quoted several writers of the seventeenth century as having mentioned aphonia occurring in the course of lead poisoning. In his exhaustive

treatise, based on observations on 1,217 cases of lead poisoning, he cited 16 patients who suffered from aphonia.

The same author mentioned the occurrence of changes in the voice and dyspnea in horses which were used at lead-works, for which tracheotomy was necessary. Tanquerel and those who preceded him represented the prelaryngoscopic era, so that observations were not made on the larynx, but their conclusions were drawn by inference from the clinical signs, such as aphonia or changes in the voice.

The number of cases of laryngeal paralysis due to lead is comparatively small, and occurrence of this condition bids fair to become even

TABLE 1.—*Cases of Laryngeal Paralyses Due to Lead Poisoning Reported in the Literature* *

Date	Author	Case	Muscles Involved
1880	Mackenzie: Die Krankheiten des Halses, Berlin, A. Hirschwald, 1880	..	Bilateral adductor
1880	Moder: Ber. d. k.k. Krankenanst. **45**: 326, 1880	..	Bilateral abductor
1881	Sajous [7]	..	Bilateral abductor
1883	Sheeh: Monatschr. f. Ohrenh. **17**: 148, 1883	..	Right unilateral abductor
1884	Seifert [8]	1	Interarytenoideus
		2	Left abductor
		3	Right abductor
1886	Remak: Berl. klin. Wchnschr. **23**: 40 (June 21) 1886	..	Bilateral abductor
1888	Kraus [4]	1	Bilateral adductor
		2	Left abductor
		3	Bilateral cricothyroid
1888	Westphal [3]	..	Bilateral adductor†
1890	Ormsby [5]	..	Aphonia; no laryngeal examination
1896	Heymann: Arch. f. Laryng. u. Rhin. **5**: 256, 1896	1	Bilateral abductor
		2	Bilateral abductor
		3	Right adductor and interarytenoideus
1897	Flatow: Deutsche med. Wchnschr. **23**: 39, 1897	..	Bilateral abductor
1905	Chodzko: Czasopismo lek. **7**: 337, 1905	..	Bilateral abductor
1907	Gelfond [6]	..	Left adductor
1909	Mosny and Stern: Bull. et mém. Soc. méd. d. hôp. de Paris **26**: 225, 1909	..	Left abductor
1913	Donelan: Proc. Roy. Soc. Med. (Sect. Laryng.) **6**: 120, 1912-1913	..	Bilateral cricothyroid
1934	Myerson	..	Right abductor

* Report of cases in the period before the use of the laryngoscope are not included.
† Seen by Kraus; probably same as that reported by Kraus.

more infrequent than heretofore because of the increased knowledge of industrial diseases and the improved methods of prevention which this knowledge has brought.

I have reviewed the literature and have found reports of 20 cases which are represented in tables 1 and 2.

The case of Westphal [3] should not be counted because this appears to be the same as the third case reported by Kraus.[4] This would reduce the number to 19. Ormsby [5] reported a case of aphonia due

3. Westphal, A.: Ueber Encephalopathia Saturnina, Arch. f. Psychiat. **19**: 620, 1888.

4. Kraus, H.: On the Disturbance of the Function of the Larynx in Diseases of the Central Nervous System, J. Laryng. & Rhin. **2**:253, 1888.

5. Ormsby, R.: Aphonia Caused by Lead Poisoning Contracted by the Abuse of Snuff, New York M. J. **52**:552, 1890.

to lead poisoning from snuff without referring to the laryngeal picture. For this reason Ormsby's case cannot be included in this study.

In some instances the larynx was involved early in the disease. This is not the rule, however. Early involvement was noted in the case of Gelfond,[6] whose patient, a woman, aged 29, became hoarse on the tenth day after using ferment tablets for making buttermilk. The tablets were packed in a lead glass jar. Analysis of the jar revealed that it weighed 190 Gm. and that 17 Gm. of its substance was lead silicate. In addition, the patient experienced colic and difficulty in moving her right arm. She recovered completely at the end of three weeks.

Differential diagnosis is not difficult as a rule, for the laryngeal paralysis is usually associated with other clinical signs or symptoms, such as wrist drop or colic. A history of exposure to lead or of an occupation as painter, potter, printer or typesetter is of value.

TABLE 2.—*Cases According to Involvement of Muscles*

Unilateral		Bilateral		Inter-arytae-noideus	Crico-thyroid
Adductor	Abductor	Adductor	Abductor		
Heymann*	Sheeh	Krause	Mader	Heymann*	Donelan
Gelfond	Seifert (2)	Mackenzie	Sajous	Seifert	Krause
	Krause		Remak		
	Mosny and Stern		Krause		
	Myerson		Flatow		
			Heymann (2)		
			Chodzko		

* Right crico-arytaenoideus lateralis and interarytaenoideus muscles.

Recovery from the laryngeal paralysis has occurred in some cases, but most authors have failed to mention this result. Sajous'[7] patient recovered on the tenth day of treatment. Seifert's[8] patient also recovered after treatment with potassium iodide.

The relative rarity of paralysis of the larynx due to poisoning by lead is apparent from a review of the literature. Prendergast[9] reported 405 cases of lead poisoning, none of which included laryngeal involvement. Relatively large series of cases of paralysis of the recurrent laryngeal nerves fail to include lead poisoning as an etiologic factor. Thus, Smith, Lambert and Wallace[10] reported 235 cases, with none

6. Gelfond, I. I.: A Case of Paralysis of the Recurrent Nerve Because of Lead Poisoning, Russk. vrach **15**:521, 1907.

7. Sajous, C. E.: Paralysis of Vocal Cord Due to Lead Poisoning, Tr. Am. Laryng. A. **3**:151, 1881.

8. Seifert, O.: Kehlkopfmuskellähmung in Folge von Bleivergiftung, Berl. klin. Wchnschr. **21**:555, 1884.

9. Prendergast, W. D.: The Classification of Symptoms of Lead Poisoning, Brit. M. J. **1**:1164 (May 14) 1910.

10. Smith, A. B.; Lambert, V. F., and Wallace, H. L.: Paralysis of the Recurrent Laryngeal Nerve, Edinburgh M. J. **40**:344, 1895.

due to lead. New and Childrey's [11] series of 217 cases and Avellis' of 150 did not include a case due to lead poisoning.

To the 19 cases just mentioned I add my own case.

REPORT OF A CASE

T. F., aged 48, a white man, was admitted to the Kings County Hospital on June 2, 1933, and died on July 25. Three weeks before admission the upper lid of his right eye began to droop, and he experienced double vision. The symptoms in the eyes increased. On the day after admission there was complete bilateral ophthalmoplegia; vision was good in both eyes. A few days later the patient experienced difficulty in swallowing. The gag reflex was abolished. He then noticed that he was unable to extend the middle, ring and index fingers of his left hand. Two days later the weakness extended to his right hand. The next development was thickness of speech.

Examination confirmed the defects just noted. In addition, the jaw deviated to the right when the mouth was opened and the uvula also deviated to the right; pharyngeal anesthesia was present, and the voice was guttural and hoarse. The triceps muscles were weak. There was marked impairment of extension of the wrists and fingers, and impairment of flexion of the left foot. On changing from a recumbent to a sitting position the head tilted backward, indicating a weakness of the muscles of the neck. It was difficult to examine the larynx with a mirror. Direct laryngoscopy revealed the right vocal cord in the midline during phonation and respiration. The patient died after fifty-three days in the hospital.

Although the larynx was removed at autopsy, histologic studies were not made on the nerves and muscles of this organ. In such cases as this, the laryngeal muscles and nerves should be studied histologically for evidence of degeneration or atrophy.

Results of the chemical examinations, by Dr. A. O. Gettler, toxicologist, are of interest: liver, 4.5 mg. of lead in 500 Gm.; intestines, 60 mg. of lead, and tibia, 6.5 mg. in 60 Gm. of bone.

A consideration of the various paralyses of the larynx due to a single etiologic factor, lead poisoning, would lead one to question the validity of Semon's law. Semon [12] postulated the theory "that the fibers of the motor nerves going to the abductors succumb to organic affections sooner than or exclusively of the adductors." He expressed the same thought in a different manner, as follows: "In all progressive organic lesions of the centers and trunks of the motor laryngeal nerves, the abductors of the vocal cords succumb much earlier than the adductors." Jeanselme and Lermoyez [13] corroborated this theory by their study, which

11. New, G. B., and Childrey, J. H.: Paralysis of the Vocal Cords: A Study of Two Hundred and Seventeen Medical Cases, Arch. Otolaryng. **16**:143 (Aug.) 1932.

12. Semon, Felix: The Study of Laryngeal Paralysis Since the Introduction of the Laryngoscope, Brain **15**:471, 1892.

13. Jeanselme, E., and Lermoyez, M.: Etude sur la contractilité postmortem, Arch. de physiol. norm. et path. **6**:109, 1885.

showed that the abductors were the first to lose their excitability after death. This observation was further corroborated by Semon and Horsley.[14] Semon further stated: "While a large number of cases of progressive organic disease of the motor laryngeal nerves have been published, in which the abductors have been shown to have undergone either alone or at any rate much more than the adductors, fatty degeneration and atrophy, not a single specimen shows the reverse." Semon had never seen a case of isolated adductor paralysis. The work of Risien Russell demonstrated that the abductor and adductor fibers of the recurrent laryngeal nerve are collected into separate bundles distinct from each other, each preserving an independent course throughout the nerve trunk to its termination in the muscle or muscles which it supplies with motor innervation. Semon believed that there was an actual biologic difference in the composition of the laryngeal muscles and nerve endings which explained the predilection of organic disease for the abductor muscles and their nerves. It is unfortunate that histologic studies were not made in the cases of adductor and interarytaenoideus paralysis of this collected series. On a clinical basis, however, there is evidence that in a series of 20 cases of involvement of the larynx due to lead poisoning, there were 2 of unilateral adductor paralysis and no other involvement. In addition, there was 1 case of unilateral adductor paralysis associated with involvement of the interarytenoideus muscle. A single case of paralysis of the interarytenoideus muscle is included, as are also 2 cases of bilateral adductor paralysis. An unusual case of involvement of both cricothyroid muscles is also included, so that 7 of 20 cases showed involvement of muscles other than the abductors. These 7 therefore constitute a definite challenge to the validity of Semon's law.

CONCLUSIONS

Paralysis of the muscles of the larynx due to lead poisoning is extremely rare.

Nineteen cases have been collected and another added. Of the 20, 7 showed paralysis of muscles other than the abductors. This would lead one to doubt the validity of the theory postulated by Semon that organic disease invariably attacks the nerves of the abductor muscles first or alone.

The general aspects of lead poisoning are discussed.

The treatment of this condition is described in standard works on the subject of lead poisoning.

136 East Sixty-Fourth Street.

14. Semon, F., and Horsley, V.: On an Apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles, Brit. M. J. **2**:405 (Aug. 28) 1886.

AGRANULOCYTIC ANGINA

FURTHER REPORT ON A CASE WITH FATAL OUTCOME FOLLOWING ORAL SURGICAL TREATMENT

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In the report in a previous issue [1] of a case of agranulocytic angina with apparent recovery, the word "apparent" was used advisedly, since it is my belief, based on the observation of a series of cases, that patients suffering from this disease, although sometimes brought back to a state of apparent good health following medication, are nevertheless always subject to recurrences if circumstances favoring such recurrences occur. The present report brings evidence to support this view.

REPORT OF A CASE

History.—Mrs. J. E. H., white, aged 52, presented at the time of her first admission on Nov. 24, 1931, the classic picture of agranulocytosis as first described by Werner Schultz, with necrotic lesions of the mucous membrane of the mouth and nasopharynx. The blood count at the height of her illness showed: white blood cells, 758; metamyelocytes, 12 per cent; staff cells, 2 per cent; segmented neutrophils, 2 per cent; lymphocytes, 80 per cent, and monocytes, 2 per cent. She received in all eighty-eight subcutaneous injections of 10 cc. each of a leukocytic extract and was dismissed from the hospital on Jan. 29, 1932, as apparently cured. Subsequent examinations made at intervals of from four to twelve weeks revealed nothing unusual in her physical condition, and she remained without complaint until the time of her last visit on Oct. 2, 1933. On that date the blood and differential counts were normal.

A report of her recent fatal illness follows. It shows dramatically the effect of an extensive surgical procedure involving the mucous membrane of the mouth, which was carried out despite the detailed and repeated warnings given her.

Fatal Illness.—The patient was admitted to Christ Hospital on June 6, 1934, complaining of generalized aching. She stated that since the time of her last visit to the office she had been free from symptoms and in perfect health. Careful questioning failed to reveal during this period any medication with the amino derivatives of the benzene group or with any other drugs. This condition of apparent perfect health had been uninterrupted until June 4, forty-eight hours before her admission to the hospital when she had had nine teeth extracted, the reason given being that the dentist thought the removal of the teeth would facilitate the proper fitting of a dental plate. On the following morning her temperature was 103 F. and she complained of marked general malaise. On the next day her temperature was 104 F. and she experienced a chill which lasted about fifteen minutes. At this time her family physician sent her from her home in a neighboring city to the hospital in Cincinnati.

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1. Bryant, B. L.: Case of Agranulocytic Angina with Apparent Recovery, Arch. Otolaryng. **16**:566 (Oct.) 1932.

Examination of her mouth at this time revealed the presence of a thick, dirty, gangrenous deposit, filling all of the sockets from which teeth had recently been removed and extending over the edges of the gums on both sides. There were no lesions elsewhere in the mouth; the tongue, buccal surfaces, pharynx, nasopharynx, hypopharynx and larynx were normal except for a moderate paling of the membranes throughout.

There was a cellulitis of the neck, and Dr. C. C. Fihe, who saw the patient in consultation, established a diagnosis of bronchopneumonia of the right upper and lower lobes, bronchitis of the left lower lobe and an external, inflamed hemorrhoid.

Smears taken from the gingival lesions at the time of the patient's admission produced on culture an abundant growth of Streptococcus viridans, Micrococcus catarrhalis and diphtheroids. Blood cultures remained sterile.

The blood counts made at the hospital are given in the table.

Subcutaneous injections of 10 cc. of leukocyte extract were given at four hour intervals, commencing immediately after the patient's admission. In addition to this two blood transfusions were given, one on June 7 at 7:30 p. m., consisting of

Counts Made Over a Period of Three Days

Date	Time	Hemoglobin, per Cent	Red Blood Cells	Color Index	White Blood Cells	Lymphocytes, per Cent	Neutrophils, per Cent	Platelets	Cells Counted
6/6/34	2:15 p. m.	73.0	3,480,000	1.05	450	100	...	108,000	50
6/7/34	7:40 a. m.	61.0	2,690,000	1.15	470	100	...	86,000	50
6/7/34	12:30 p. m.	....	2,910,000	....	...	...	...	.....	..
6/7/34	4.00 p. m.	60.0	2,760,000	1.07	427	100	...	.....	50
6/8/34	7:20 a. m.	....	.....	....	200	100	...	.....	25
6/8/34	4:00 p. m.	69.0	3,410,000	1.00	290	96	4.0	.....	25

280 cc. of citrated blood and 200 cc. of a physiologic solution of sodium chloride, and the second on June 8 at 1:15 p. m., consisting of 500 cc. of citrated blood and 200 cc. of physiologic solution of sodium chloride.

The patient's temperature on admission was 104.2 F. and ranged thereafter between 101.6 F. and 106 F. On June 8, the second day after admission, there developed an indurated, red area 2 inches (5 cm.) wide and 3½ inches (8.9 cm.) long on the right buttock, beginning in the region of the infected hemorrhoid. That evening the patient became delirious; the following morning, June 9, she was comatose and died at 9:50 a. m.

One hour after death a necropsy was performed which was supplemented by the microscopic examination of specimens of tissue from the various organs of the body. No detailed report is presented here, but the report of the pathologist, Dr. D. B. Remson, on the macroscopic and microscopic examination of the spleen and bones follows:

Macroscopic Observations.—Spleen: The spleen was not enlarged. The capsule was blue-gray and appeared a little thickened. On section the malpighian bodies were seen distinctly, and the pulp had a slightly dull gray cast. The trabeculae were unusually prominent. The vessels were not remarkable. Bones: A small window was made in the femur. The marrow appeared fatty and partially liquefied. Blocks were taken from several ribs, the manubrium and one of the vertebrae. All of these showed an apparent aplasia of the bone marrow. The bony trabeculae stood out in such a way as to suggest the appearance of a dried sponge.

Microscopic Observations.—Spleen: The section showed a normal capsule. The substance itself had a curiously compact appearance difficult to analyze. The malpighian bodies appeared normal. The sinuses were compressed and the cells lining them appeared larger than usual. There was some blood in the pulp, but most numerous were the clear splenic cells and cells with darker staining, round nuclei. Many scattered, early marrow cells were seen. No granulocytes were visible. The diffuse pink staining through the pulp did not clearly suggest an increase in connective tissue.

Marrow of the Femur: The section showed considerable hemorrhage, but the marrow cells were extremely scarce. A few megaloblasts were recognizable, but myeloid cells were rare.

Ribs and Vertebrae: The sections showed a normal bony structure but the marrow in each was hypoplastic. The process of decalcification of the tissue had made an analysis of the cells unreliable, but it appeared that such myeloid cells as were present represented very early forms.

SUMMARY

It was deemed advisable to present this report for two reasons: First, the patient's previous illness had formerly been reported as apparently cured. Second, it is impossible to impress too strongly on patients who have had this disease and on the physicians in attendance the importance of avoiding rigorously, surgical procedures which concern the mucous membranes of the body; further, the importance of carefully attending to and treating any spontaneous lesions of the membranes which may occur, no matter how insignificant they may be, and finally, the importance of maintaining a careful, regular and more or less permanent check-up with complete examinations of the blood of patients known to have suffered from granulopenia. If surgical treatment involving the mucous membranes is at any time urgently indicated in such a case, the patient should be studied carefully before the operation and prepared by injections of leukocyte extract, pentnucleotide or some similarly acting substance, so that the extra drain on the part of the hematopoietic system concerned with the production of the granulocytes can be compensated for by the stimulation of these remedial agents, which may be expected to act prophylactically.

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HAY FEVER AMONG JAPANESE

PART I

H. J. HARA, M.D.
LOS ANGELES

A number of leading otolaryngologists in Japan, Korea and Manchuria have stated in personal communications that hay fever is practically unknown among the natives. On American soil, however, where hay fever is endemic, it appears to develop in the same racial stock. During the past five years I have seen and treated scores of Japanese patients with hay fever in Southern California, in both private and dispensary practice. To what extent hay fever may be present among this race has been largely a matter of conjecture. No contribution has appeared in the literature on this subject. To solve this apparent contradiction as to racial immunity, a survey was conducted in a given area containing a sufficient number of persons suffering from hay fever. I believe that such a study may open a new avenue through which an approach may be made for future investigation on the problem of allergy, the solution of which would determine the rational therapeutic measures to be instituted.

The survey was begun in November 1931 and lasted several months. The territory covered was 731 square miles, corresponding to the three health districts which are now under the supervision of the Los Angeles County Health Department. This included all the San Gabriel and Pomona Valleys and the mountain region in the north to the Sierra range; it extended east to the western boundary line of San Bernardino County and south to the boundary of Orange County. The western boundary was San Gabriel Boulevard, running north and south. The cities of Sierra Madre, Monrovia, Azusa, Pomona, Whittier, Montebello and Norwalk and also the surrounding nonincorporated towns are located within this area (shaded area of the map). This region was selected because of its proximity to the city of Los Angeles and because a large number of Japanese reside here. According to the statistics of the Los Angeles County Health Department of June 1931, there was a total population of 230,000. Dr. John L. Pomeroy, county health officer and professor of public health of the College of Medical Evangelists, assisted me in carrying on the study as part of the regular public health program in the district. Through the Japanese Chamber of Commerce of El Monte, the names and addresses of each family in the district were obtained. The house to house canvass was done by a Japanese medical student under the County Health Department. When a suspected or known case of hay fever was encountered I interviewed the subject at length. The investigation covered the items listed in table 1.

This article (part I of thesis work) was submitted to the Faculty of Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Doctor of Medical Science (D.Sc. [Med.]) for graduate work in otolaryngology.

After several months of intensive effort during which I and my co-workers often worked late at night in order to interview those who were away from their homes during the day, 459 families were canvassed, and first hand information was obtained from 1,937 persons. Figure 2 indicates their age, sex and place of nativity. There were 1,118 American-born Japanese, 16 Hawaiian-born and 803 Japanese-born. In looking over the chart, the student of sociology can readily recognize the effect of the recently enacted immigration ban on the Japanese, as indicated by the comparatively small number of young adults now residing in this country. There is also a wide gap in the age distribution between the first and second generations.

TABLE 1.—*Questionnaire Used in the Survey*

1. Name and address	Age	Married Widowed	Occupation
	Sex	Single	Phone
2. Address in Japan			
3. Family	Husband	Age	Siblings
	Wife	Age	
4. How many years in United States?		In present location?	
5. Occupation in Japan			
6. Does or did your near relative ever suffer from			
(a) Hay fever?		Which relative?	
(b) Asthma?		Which relative?	
(c) Urticaria?		Which relative?	
(d) Eczema?		Which relative?	
7. Do or did you ever suffer from hay fever? (a) Yes (b) No			
If yes, describe as to:			
Onset and duration		Where?	
Jan. Feb. March April May June July Aug. Sept. Oct. Nov. Dec.			
When worse?		What substances?	
What previous treatments, if any?			
What nasal operation, if any?		For what purpose?	

A similar survey was undertaken among the Japanese population in the city of Los Angeles. Several months were spent in making this study. Owing to the transient state of many of the persons and the chances of missing a case here and there, the work was difficult. Many data were obtained, but I believe that they do not approach in accuracy those from the country district, where the population is more stable. For this reason no figures for the city are presented here.

Among the 1,937 persons interviewed, 30 gave a positive history of hay fever of varying duration or clinical symptoms characteristic of hay fever. There were 28 adults born in Japan, 1 adult born in Hawaii and 1 boy, aged 10, born in America. Sixteen submitted to the scratch test, 12 of whom took a series of treatments of varying duration.

Fig. 1.—Map showing territory in which survey was made (shaded portion).

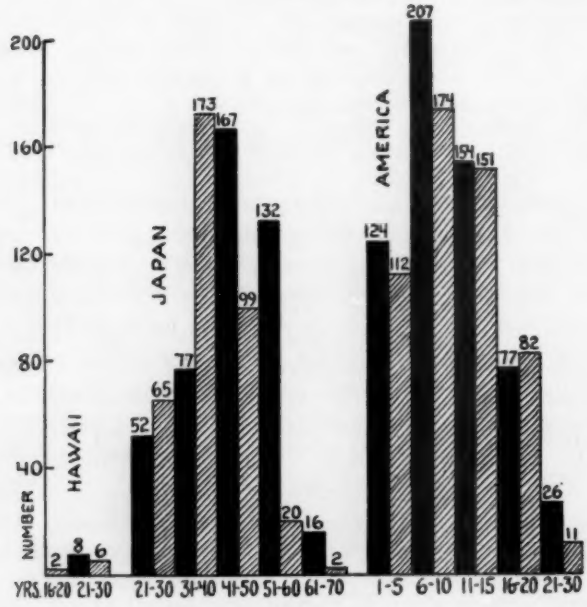

Fig. 2.—Distribution of 1,937 persons for whom data was obtained according to age, sex and land of nativity; the solid black sections indicate males and the sections with diagonal lines females.

During the season of 1932, 25 others not included in these data were seen, and a positive diagnosis was made. These subjects took a series of pollen treatments. These included 2 children under 10 years of age, who were born in America; 1 child born in Hawaii; 1 adult who had suffered from hay fever in Hawaii fifteen years before and who later came to America, and 21 adults born in Japan. These patients cooperated well and made possible laboratory and clinical examinations at regular intervals, including the time beyond the close of the desensitization period in 1932 and during 1933.

Pollen extracts for diagnosis and treatment were furnished by Dr. Alfred R. Roos of the department of immunology of the College of Medical Evangelists. Diagnostic scratch and intradermal tests were conducted by me and my office assistants, and the reaction noted was that of the generally accepted standard used by allergists in this country.

Figure 3 indicates the monthly period of sensitivity of these 55 patients classified in three groups. The dark areas indicate the months in which the subjective symptoms were most severe; the shaded areas indicate the months in which they were moderately severe, and the light areas the months in which the patients were free from hay fever. Group A is comprised of 14 patients in the surveyed area who had definite subjective symptoms of sensitivity; group B consists of 16 patients from the surveyed area who submitted to diagnostic skin tests, and group C is made up of 25 patients in an area outside that of the survey whose condition was diagnosed and who took treatments.

The first 18 patients listed did not take the pollen treatments. Of the remaining 37, 7 took one or more series of treatments before the season of 1932. Of the remaining, 30 took treatments for the first time. Eight of these stopped before completing the series, owing to lack of understanding of the problem or failure to keep appointments because of distance. Nearly all the treatments were coseasonal and consisted of from thirty to forty injections which were given two or three times weekly, depending on the individual reaction. During the winter of 1932 these cases were followed up, and each patient was advised to take preseasonal treatments the following year. Only 2 patients (33 and 34) returned for preseasonal treatment; the remainder waited until symptoms appeared and then took another series of coseasonal treatments. Generally, the season of 1933 was much easier for the majority of the patients. With the exception of patients 34 and 50, who felt much better after the treatments taken in 1932 but who were worse during the season of 1933, most of the subjects required a fewer number of injections than during the previous year.

Another group of about 30 patients began their treatments for the first time in 1933. This period of observation being limited to one season, they are not included in this study.

Table 2 presents various items of interest.

ANALYSIS OF FIFTY-FIVE CASES OF HAY FEVER

There were 15 farmers, 10 gardeners, 9 florists, 9 housewives and 3 school children in the group treated. The other patients were teachers, housemen, clerks, book-keepers, etc. The majority of farmers, gardeners and florists in this group can be explained by the fact that they were constantly exposed to pollens throughout the year.

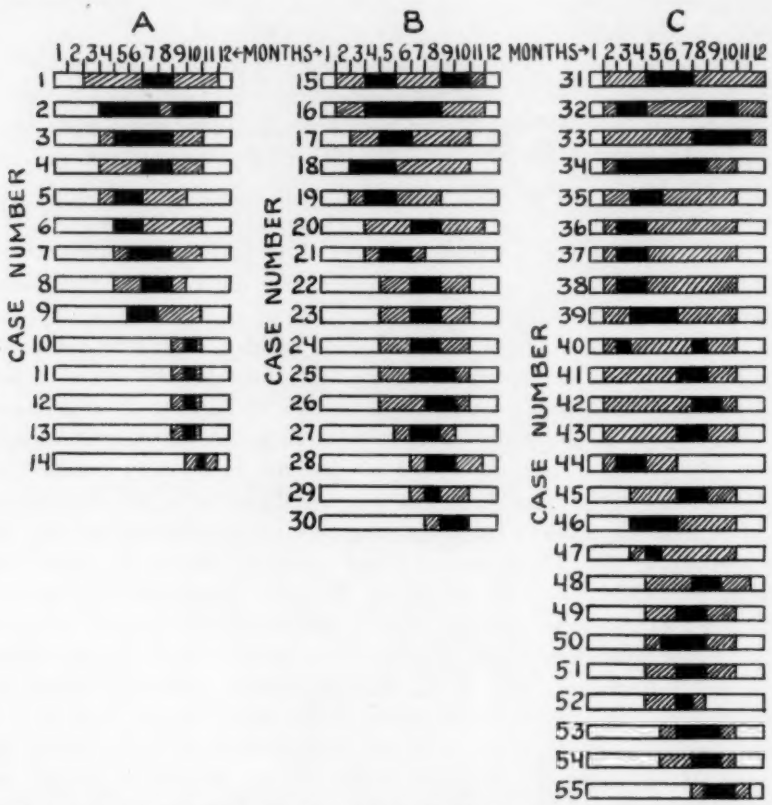

Fig. 3.—Distribution of the 55 patients who received pollen treatment according to the monthly period of sensitivity. Group *A* consists of 14 patients in the area of the survey who had definite subjective symptoms of sensitivity; Group *B* consists of 16 patients in the area of the survey who submitted to diagnostic skin tests. Group *C* is composed of 25 patients in an area outside that of the survey whose conditions were diagnosed and who took treatments. The solid black sections indicate the month in which the subjective symptoms were most severe, the sections with diagonal lines those in which they were moderately severe and the light sections those in which the patients were free from hay fever.

All but 4 patients were natives of Japan; 1 was from Hawaii, and 3 were children born in America of Japanese parentage.

TABLE 2.—*Data for Twenty-Five of the Fifty-Five Patients Who Received Pollen Treatments*

TABLE 2.—*Data for Twenty-Five of the Fifty-Five Patients Who Received Pollen Treatments*

Case	Age	Sex	Occupation	Married or Single	Land of Nativity	Years in United States	Years in Present Locality	Duration of Hay Fever	Family History	Other Allergies	Previous Pollen Treatments	Evidence of Focal Infection	Previous Nasal Operations	Results of Treatment
31	36	F	Housewife	M	Japan	14	12	4	Negative	None	None	None	Caldwell-Luc operation	Fair
32	42	M	Teacher	M	Japan	19	9	9	Negative	Asthma	2 years ago	None	Turbinectomy	Good
33	27	M	Clerk	M	Japan	11	11	4	Negative	None	None	None	None	Poor
34	40	M	Janitor	M	Japan	Hawaii, 15; United States, 10	10	15	Negative	None	1 year	None	Caldwell-Luc operation	Poor
35	29	M	Gardener	M	Japan	14	7	1	Negative	None	None	Chronic follicular tonsillitis	None	Good
36	26	M	Gardener	S	Japan	7	1	1	Negative	None	None	None	Intranasal antrum window	Good
37	23	F	Housewife	M	Japan	4	4	2	Negative	None	None	None	None	Good
38	39	F	Housewife	M	Japan	14	8	2	Negative	None	2 years ago	Chronic follicular tonsillitis	None	Good
39	21	M	Gardener	S	Japan	11	5	3	Negative	None	3d year	None	None	Condition much better
40	35	M	Gardener	M	Japan	25	10	5	Negative	None	None	None	Polypoidectomy	Fair
41	30	M	Chauffeur	M	Japan	12	1	1	Negative	None	None	None	None	Good
42	46	F	Housewife	M	Japan	9	9	1	Negative	None	None	Chronic follicular tonsillitis	None	Poor
43	29	M	Clerk	S	Japan	6	4	3	Negative	None	None	None	Submucous resection	Fair
44	62	M	Gardener	M	Japan	40	10	18	Negative	Asthma	1 year	None	Polypoidectomy	Fair
45	10	F	School child	S	Hawaii	10	10	3	Negative	Asthma	1 year	None	.....	Good
46	29	F	Housewife	M	Japan	8	8	2	Negative	None	None	Chronic follicular tonsillitis	None	Good
47	33	F	Housewife	M	Japan	10	10	1	Negative	None	None	None	None	Good
48	26	M	Houseman	S	Japan	13	2	2	Negative	None	None	None	None	Good
49	28	F	Housewife	M	Japan	6	6	3	Father asthmatic	None	None	None	None	Fair
50	59	M	Janitor	M	Japan	17	5	6	Negative	None	None	Pyorrhea	Polypoidectomy	Good
51	59	M	Teacher	S	Japan	25	20	2	Negative	Asthma 43 years	None	None	None	Fair
52	8	M	School child	S	United States	8	7	7	Uncle asthmatic; mother had hay fever	None	None	None	None	Good
53	51	M	Farmer	M	Japan	20	12	10	Negative	None	None	Chronic follicular tonsillitis	None	Not given
54	42	M	Gardener	M	Japan	15	7	2	Negative	None	None	None	Polypoidectomy	Fair
55	48	F	Housewife	M	Japan	20	10	16	Negative	None	None	None	None	Fair

There were 35 males and 20 females, making the proportion about 5 males to 3 females. Males were in the majority probably because of their greater opportunity for exposure to pollen-laden air.

There were 3 patients under 10 years of age, 16 in the third decade, 13 in the fourth, 18 in the fifth and only 5 in the sixth. It would appear that hay fever spares no age group, but occurs most frequently during adult life.

Figure 4 groups patients according to the onset of hay fever subsequent to their coming to America, but not including the Hawaiian-born and American-born. It is interesting that a race whose forefathers were apparently free from a disease for centuries can contract it in a new environment when the required conditions are fulfilled. It would appear that the process of sensitization is slow and insidious. The sensitized patients must have been subjected to repeated exposure to relatively large doses of irritating pollen. In some instances the allergic soil must have been in a state of cultivation for years before the seeds fallen on it germinated, budded, bloomed and came to fruition.

Fig. 4.—Relation of length of residence in the United States to the development of hay fever; the solid black sections indicate males and the dotted sections females.

It is to be noted that in all the females subjective symptoms developed before the twelfth year of their residence in America. Among the males, symptoms developed in 16 of 35 after the thirteenth year, as shown in the right half of the chart. Therefore, males appear to have a greater resistance to sensitization to pollen.

Forty-seven patients gave an irrelevant family history. Eight, or 14 per cent, gave a definite history of allergy in immediate blood relatives. Two had asthmatic fathers, 1 of whom (patient 21) also had hay fever. One had an asthmatic mother; 2 had asthmatic uncles, and 1 had an asthmatic great-grandmother. One had a mother with hay fever, who was treated in 1933, and 1 an asthmatic brother. Doubtless, many failed to give an accurate history of family antecedents because of their absence from near relatives in their native land; therefore, a negative history meant little.

Four of this group suffered from asthma. None had dermatologic, gastro-intestinal or neurologic symptoms.

Nine reported having had various types of nasal operations performed on one or more occasions. These included four polypoidecto-

mies, two Caldwell-Luc operations, one submucous resection, one turbinectomy and one operation on the intranasal antrum with window resection. All the patients had their nasal operations following the onset of hay fever, and none obtained any perceptible relief from the hay fever. One (patient 28) stated that the operation made his nose worse. Unquestionably, the pathologic condition of the nose was not the cause of the hay fever, but was coincidental.

ASTHMA AND ALLERGIC CONDITIONS IN JAPAN

Asthma is prevalent in Japan. In a personal communication, Prof. Dr. Ino Kubo gives the following report, which was taken from the records of the outpatient department of the University Hospital at Fukuoka:

1929	Total number admitted to nose and throat room....	5,111
	Number of cases of asthma.....	62
	Males, 44; females, 18	
1930	Total number admitted to nose and throat room....	4,631
	Number of cases of asthma.....	53
	Males, 33; females, 20	

Of the 115 asthmatic patients who were treated, 2 had asthmatic mothers; 1 had 3 brothers, all of whom were asthmatic, and another had a mother whose sister was asthmatic. There were 5 asthmatic patients whose parents were near blood relatives. The majority of these 115 patients had their worse attacks during the winter. Three cases of chronic vasomotor rhinitis were complicated with asthma. The relative prevalence of bronchial asthma during the winter in Japan would therefore seem to indicate its nonpollen origin.

An interesting observation has been made regarding the freedom of those suffering from hay fever from their usual seasonal symptoms during their stay in Japan. Dr. K. I., a Japanese and a graduate of Stanford University School of Medicine, who practiced medicine in Los Angeles for twenty years, has suffered from hay fever complicated with hyperplastic rhinitis. Recently he visited Japan, staying there nearly a year, and while there was completely free from symptoms. On his return to America his nose felt normal and acted normally. There was no evidence of polyposis. This continued for about a year; then gradually the usual symptoms reappeared. An American-born girl, aged 11, stated that her symptoms persisted even during her twenty-four hour stop-over in Honolulu, but that a few hours after leaving the shores of Hawaii and through all her journey in Japan she was perfectly well and remained well until a year after her return to America. Three patients in this group and scores of other patients with hay fever who went to Japan and later returned to this country passed through similar experiences.

I am not unmindful of the splendid contribution on the relation of a diet high in protein and allergy, nor can I fail to recognize the claims of the champions of a diet high in carbohydrate as the possible source of allergy. Experience, however, has shown that one's dietary habits remain practically the same, regardless of where one lives. The Japanese are no exception. As a race, they thrive on a diet low in protein and high in carbohydrate. Those in whom hay fever developed in America and who remained free from symptoms during their visit in Japan lived there much as they did in America. The evidence at hand would seem to indicate that diet plays but little part in the pathogenesis of hay fever.

COMMENT ON SURVEY AND CLINICAL STUDY

From the evidence presented, one can safely state that the Japanese show no racial immunity to hay fever, either natural or acquired. The incidence of hay fever among this people is about 3.5 per cent of the population in the suburban and rural territories in Southern California. As in other races, a hereditary atopic background plays a part. The ratio between male and female is 5 : 3; the female shows much less resistance to pollens, as shown by the appearance of symptoms much sooner among females than among males after coming to this country. In all these patients the symptoms of hay fever developed in Southern California. Dietary habits play no part in the production of hay fever.

AURAL TUBERCULOSIS

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More than a century before 1882, when Koch discovered the tubercle bacillus, a French surgeon, Jean Louis Petit, who lived between 1674 and 1750, described tuberculosis of the mastoid. His observations were followed by those of Romberg, Griselle, Nelaton, Rilliet, Barthez and Geissler, who mentioned the connection between tuberculous dyscrasia and tuberculosis of the middle ear and mastoid.[1] Many other valuable anatomic and clinical contributions followed, among them those of Wilde,[2] Politzer,[3] Schwartze [4] and others, so that at the time the causative organism of tuberculosis was demonstrated the definite clinical entity of aural tuberculosis had been established. Considerable impetus was given to the study of the histologic pathology by Koch's discovery as evidenced by the writings of well known otologists of that time, Habermann,[4] Schwabach,[4] Korner,[4] Hegetschweiler,[4] Barnick [4] and others.

In 1883 Esche [5] first actually demonstrated the presence of the tubercle bacillus in the secretion from the middle ear of tuberculous patients. Later Nathan [6] stated that he always found the tubercle bacillus in the secretion from the middle ear of phthisical patients, but his statement was soon contradicted, notably by Schwabach and Brieger [4] who declared, however, that the inability to find the bacillus did not necessarily mean that the aural condition was not tuberculous.

In the early work on tuberculous otitis media considerable emphasis was laid on the destruction of tissue taking place in the middle ear and mastoid as the result of the tuberculous infection. It was frequently

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Candidate's thesis read before the fortieth annual meeting of the American Laryngological, Rhinological and Otological Society, Charleston, S. C., April 4, 1934.

1. Ormerod, F. C.: J. Laryng. & Otol. **46**:449 (July) 1931.
2. Wilde, W. R. W.: Practical Observations on Aural Surgery, London, John Churchill, 1853.
3. Politzer, Adam: A Text-Book of the Diseases of the Ear, for Students and Practitioners, ed. 5, London, Baillière, Tindall & Cox, 1909.
4. Quoted by Politzer.[3]
5. Esche: Tuberkelbacillen in dem Ausflusse bei Mittelohreiterungen von Phthisikern, Deutsche med. Wchnschr. **9**:441, 1883.
6. Nathan: Deutsches Arch. f. klin. Med. **35**:491, 1884.

alluded to as a terminal condition, and the prognosis was almost universally considered grave.

In 1900 Seymour Oppenheimer [7] stated that the ossicles were frequently entirely destroyed, or dislocated and came out. He called attention to the fact that 7 cases of tuberculous otitis in which death resulted from ulceration of the carotid artery had been reported.

In 1902 Grimmer [8] reported several cases among children, many of whom had severe involvement of the temporal bone. He included reports of cases in a few adults, 2 of whom had cholesteatoma and 1 chronic purulent otitis media followed by acute mastoiditis with Bezold's perforation. No laboratory evidence was brought forth to prove the presence of tuberculosis of the ear in these adults.

In 1903 Milligan [9] noted that tuberculous otitis media was most frequent among infants and young children. He believed that primary tuberculosis was probably more frequently encountered than was usually supposed.

In the same year Goldstein [10] reported 4 cases of primary tuberculous otitis media and mastoiditis, 2 of which were in adults. A few similar isolated cases had been reported prior to that time, 1 by Herman Knapp [11] in 1894, 1 by Williams [12] in 1895, 1 by McCaw [13] in 1901 and 1 by Jobson Horne [14] in 1903. Hurd [15] and Oppikoffer [16] each reported 1 case in 1905.

In 1903, Levy [17] reported that practically all of his cases were secondary and of a mild nature, without an unfavorable prognosis. He attributed this to the fact that they occurred mostly in adults.

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7. Oppenheimer, Seymour: The Pathology, Diagnosis, Prophylaxis, and Treatment of Tuberculosis of the Middle Ear, Ann. Otol., Rhin. & Laryng. **60**: 397, 1900.

8. Grimmer, G. K.: A Contribution to the Pathology and Diagnosis of Tuberculous Otitis Media, Arch. Otolaryng. **31**:181, 1902.

9. Milligan, W.: Diagnosis and Treatment of Tuberculous Disease of the Middle Ear and Accessory Cavities, J. Laryng., Rhin. & Otol. **18**:137, 1903.

10. Goldstein, M. A.: Primary Tuberculosis of the Middle Ear and Mastoid, J. Laryng., Rhin. & Otol. **18**:113 (March) 1903.

11. Knapp, Herman: A Case of Primary Tuberculosis of the Mastoid Process, Arch. Otolaryng. **23**:64, 1894.

12. Williams: Tr. Minnesota M. Soc. 1895, p. 161.

13. McCaw, J. F.: M. News (Oct. 12) 1901.

14. Horne, W. Jobson: Primary Tuberculosis of the Ear, J. Laryng. Rhin. & Otol. **18**:197 (April) 1903.

15. Hurd, L. M.: Primary Tuberculosis of the Ear, Arch. Otolaryng. **34**:311 (Aug.) 1905.

16. Oppikoffer, E.: Chronic Purulent Otitis Media on the Left Side with Empyema of the Mastoid Process and Necrosis of the Facial Nerve, Arch. Otolaryng. **34**:290 (Aug.) 1905.

17. Levy, R.: Middle Ear Disease in Tuberculosis, Laryngoscope **13**:357, 1903.

It is possible that tuberculosis of the middle ear as seen in the present day is not so severe an infection as it formerly was. No less an authority than Sir St. Clair Thomson [18] recently declared "I have never observed caries, discharge of the ossicles, facial paralysis or mastoid tenderness, nor have I seen any extension to the temporal bone, lateral sinus, meninges, or brain." He also had seen relatively few cases in which there were multiple perforations of the membrana tympani.

In the series covered by this report I saw no striking evidence of the destructive action of the tubercle bacillus in the ear or mastoid of adults. In fact, several cases were observed over a period of weeks with no appreciable change in the aural condition. In 2 suspected cases the patients never had any drainage from the ear, and 1 of them showed a definite improvement in both the aural and the pulmonary lesion before leaving the hospital. In several other cases scant draining continued, but there was no evidence of any increase in the size of the small pinpoint perforation, nor were there any new perforations.

INCIDENCE OF TUBERCULOUS OTITIS MEDIA IN RELATION TO OTHER FORMS OF TUBERCULOSIS AND TO CHRONIC PURULENT OTITIS MEDIA

In the study of these cases an attempt was made to determine the incidence of tuberculous otitis media in adults and its relation to other forms of tuberculosis and to chronic purulent otitis media. The clinical course of tuberculous otitis media was also followed so far as circumstances permitted when the hospital residence of each patient was short. No serious attempt at treatment was made because of inability to follow the cases.

As conservative opinion seems to be that the most reliable methods of proving the presence of tuberculosis are inoculation of animals with suspected material and the microscopic examination of granulations removed from the ear or mastoid, only a relatively small number of cases were examined in the laboratory by methods other than these. The long delay after inoculation of animals is often cited as an objection to this method of arriving at a diagnosis, but I do not believe that it is a serious drawback in adult aural tuberculosis, since these cases, as a rule, progress slowly, if at all.

I do not believe that the diagnosis can be definitely arrived at clinically. There are many typical cases, but there are also cases, shown by inoculations of animals to be tuberculous, which are atypical. Nor do I believe that negative results from inoculations of animals are infallible, for many cases in which there was a typical history with a suspicious

18. Thomson, St. Clair: J. Laryng. & Otol. **46**:460 (July) 1931.

aural condition did not show tuberculosis, and in 2 cases in which the aural exudate was injected into animals monthly, positive and negative laboratory findings were reversed from one month to the next.

A total of 974 patients with pulmonary tuberculosis, ranging from the incipient to the far advanced form with tuberculosis of other organs complicating the original lesions, was examined. There were 45 cases of chronic purulent otitis media, 12 of which were proved to be caused by the tubercle bacillus, and 5 of which were considered positive because of the history of onset, the appearance of the membrana tympani and the clinical course of the disease, although the laboratory observations were negative. Four and seven-tenths per cent of the patients had chronic discharging ears, 1.7 per cent tuberculous otitis media (1.2 per cent substantiated by laboratory studies) and 3 per cent simple chronic purulent otitis media.

A comparison of these findings with those of other observers is of interest.

TABLE 1.—*Incidence of Aural Infections in Patients with Chronic Pulmonary Tuberculosis*

Total number of patients examined	974
Total number of patients with aural discharge	45
Number of proved cases of tuberculous otitis media	12
Number of suspected cases of tuberculous otitis media	5
Number of cases of simple chronic purulent otitis media	28

Cemach [19] reported 4.7 per cent of all his cases of chronic purulent otitis media as tuberculous, but this figure rose to 14 per cent in children. Of the patients with otorrhea requiring operation 8 per cent were tuberculous and 22 per cent of the phthisical patients with suppurating ears were tuberculous.

Chapman [20] said there are from 4 to 6 cases of aural tuberculosis in each 100 cases of pulmonary tuberculosis.

From 1920 to 1929 at Brompton Hospital Ormerod [1] found 58 cases of tuberculous otitis media in a series of 11,384 cases of suspected pulmonary tuberculosis, 50 per cent of which were probably tuberculous. This shows an incidence of about 1 per cent. In table 2 he compares his findings with those of other observers.

Collins [21] found 4 cases of tuberculous otitis media among 62 cases of advanced chronic pulmonary tuberculosis. One of these showed a bilateral involvement.

19. Cemach, quoted by Ormerod.[1]

20. Chapman, S. J.: Tuberculosis of the Middle Ear with Especial Reference to Heliotherapy, Ann. Otol., Rhin. & Laryng. **36**:631, 1927.

21. Collins, B. C.: The Middle Ear in Tuberculosis, Brooklyn M. J. **15**:595, 1901.

It will be noted that in these reports which apparently concern adults, the tuberculous otitis either accompanied or followed chronic pulmonary tuberculosis.

The statistics for infants and children are different. Ormerod [1] said that secondary tuberculosis is almost invariably seen in adults, while in infants the disease is frequently apparently primary.

At necropsy of 120 tuberculous children Carr [22] found 3 cases of tuberculous involvement of the ear and temporal bone.

In 21 necropsies on children Price-Jones [23] found tuberculous otitis in 2.

Still [24] in 269 necropsies on children with tuberculosis found tuberculous otitis in 15.

Mathews [25] found tuberculous involvement of the middle ear in 7 cases, 2 unsuspected, in 25 necropsies on children. He believes that in children up to 3 or 4 years of age tuberculous otitis is primary.

TABLE 2.—*Incidence of Various Forms of Tuberculosis*

Observer	Number of Cases of Pulmonary Tuberculosis Examined	Cases of Laryngeal Tuberculosis	Cases of Tuberculous Otitis	Cases of Nontuberculous Otitis	Cases of Tuberculosis of the External Canal
Szinetl	400	.....	34	(8.5%)	..
Cemach	2,342	.....	56	(2.4%)	..
Moldenhauer	294	.....	7	(2.4%)	..
Schwabach	113	.....	8	(6.9%)	..
Ormerod	11,384*	2,119	58	(1.0%) 321	1

* Fifty per cent considered tuberculous.

In 100 consecutive fatal cases of disease of the middle ear Whitehead [26] found 12 cases of aural tuberculosis, 9 of which were in children under 2 years of age. In 8 of these the condition appeared to be primary.

In general these findings are substantiated by many observers, including Milligan,[9] Turner and Fraser,[27] Briggs,[28] McCart,[29] Cox and Dwyer [30] and others.

22. Carr, cited by Mathews, P.: Brit. J. Child. Dis. **3**:423 (Oct.) 1906.

23. Price-Jones, C.: Causation of Tuberculosis, Practitioner **71**:191, 1903.

24. Still, G. J.: Morbid Anatomy of Tuberculosis, Brit. M. J. **2**:455 (Aug. 19) 1899.

25. Mathews, Paul: Tuberculosis of the Middle Ear in Children with Especial Reference to Its Occurrence as a Primary Lesion, Brit. J. Child. Dis. **3**:423 (Oct.) 1906.

26. Whitehead, A. L.: J. Laryng., Rhin. & Otol. **18**:158 (March) 1903.

27. Turner, A. L., and Fraser, J. S.: Tuberculosis of the Middle Ear Cleft in Children. Clinical and Pathological Study, J. Laryng. & Otol. **30**:209, 1915.

28. Briggs, H. H.: Tuberculosis of the Middle Ear, Ann. Otol., Rhin. & Laryng. **23**:529, 923, 1914.

29. McCart, H. D.: Tuberculosis of the Middle Ear, J. Laryng. & Otol. **40**: 451 (July) 1925.

30. Cox, G. H., and Dwyer, J. G.: Tuberculosis of the Middle Ear, Ann. Otol., Rhin. & Laryng. **24**:371, 1915; Arch. Otolaryng. **9**:414 (April) 1929.

It appears that tuberculous otitis is a serious disease among infants and children, that the condition is acute with severe complications and an unfavorable outlook. Also among children a considerable number of cases of chronic purulent otitis media, not suspected of being tuberculous, have proved to be tuberculous. The studies of Cox and Dwyer [30] have recently shown that over 15 per cent of all the cases of chronic purulent otitis media in children are of tuberculous origin, the bovine type of organism being most frequently found.

Briggs [28] said: "In children the process is liable to be acute and extensive with much breaking-down of temporal bone. In adults the pathological changes are limited to the middle ear and are chronic from the onset."

One hundred and twenty cases of chronic purulent otitis media were studied. Of these 102 occurred in patients who had chronic pulmonary tuberculosis. Thirteen subjects were indefinitely nontuberculous, and 5 patients had tuberculosis of the bones and joints.

TABLE 3.—*Incidence of Tuberculous Otitis in Patients with Chronic Pulmonary Tuberculosis and Chronic Aural Discharge*

Total number of cases with otorrhea and chronic pulmonary tuberculosis..	102
Number of cases in this group with negative laboratory evidence (of this number 18 were considered clinically positive).....	69
Number of cases in this group with positive laboratory evidence.....	33

Of the 102 cases of chronic pulmonary tuberculosis, 69 did not have laboratory evidence of tuberculous otitis media. There were 18 in the group which were considered as clinically positive, but in which definite laboratory evidence of tuberculosis of the ear could not be obtained even after repeated inoculations of animals. Of the 69 cases, 48 showed positive evidence of tuberculosis in the sputum. Seventeen gave negative results on repeated examination of concentrated sputum; 3 showed questionable, probably positive, results, and in 1 no sputum was obtainable.

Thirty-three cases showed definite positive laboratory evidence of tuberculous otitis media, a total of about 33 per cent of all the chronic purulent cases of otitis media occurring in tuberculous persons.

In a series of 41 cases of chronic pulmonary tuberculosis and chronic purulent otitis media reported by Lussmann and Bendove,[31] tubercle bacilli were found in the aural exudate in 7, or 17 per cent.

Thirteen definitely nontuberculous persons, with chronic purulent otitis media on whom an operative procedure (simple, modified radical or radical mastoidectomy) was used were examined, and none of them

31. Lussmann, W. J., and Bendove, R. A.: Chronic Otitis Media in the Tuberculous, Arch. Otolaryng. **6**:153 (Aug.) 1927.

showed evidence of tuberculous infection of the ear or mastoid as demonstrated by the microscopic sections of granulations removed from the middle ear and mastoid at operation, and by inoculations of animals with purulent discharge from the ear.

Five cases of chronic purulent otitis media were seen among patients with tuberculosis of the bone. None of them showed evidence of tuberculous otitis media. One of these patients who had tuberculosis of the left hip of two years' and chronic purulent otitis media of seven years' duration died of tuberculous meningitis. Necropsy confirmed this diagnosis and showed no evidence of tuberculosis in the temporal bones.

Age Incidence.—The outstanding observation was that tuberculous otitis media is much more common among young adults. The average age of all the patients in the series was 32 years. The average age of patients with tuberculous otitis media was 28 years, while the average age of those with pulmonary tuberculosis and chronic purulent otitis media was nearly 35 years. The youngest patient with tuberculous otitis was 18; the oldest, 59.

TABLE 4.—*Age Incidence*

	Age in Decades								
	20 to 30		30 to 40		40 to 50		50 to 60		
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Totals
Negative	25	38	19	29	13	20	9	14	66
Positive	22	66	9	27	1	3	1	3	33

Of 33 patients with tuberculous otitis media, 22 (66 per cent) were between 20 and 30 years of age; of 66 with chronic purulent otitis media, 25 (38 per cent) were in the same decade. Nine patients with tuberculous otitis (27 per cent) were between 30 and 40; 19 with chronic purulent otitis media (29 per cent) were in the same age group. One patient with tuberculous otitis (3 per cent) was between 40 and 50, and 13 with chronic purulent otitis media (20 per cent) were in the same age group. There was 1 case of tuberculous otitis media (3 per cent) in the group aged from 50 to 60 years, and there were 9 cases (14 per cent) of chronic purulent otitis media. No patient was over 60, and 5 who were included in the group from 20 to 30 years of age were under 20.

Five patients with tuberculous otitis media were Negroes, while there was only 1 Negro in the group with a diagnosis of simple chronic purulent otitis media.

Sex Incidence.—There were 14 females and 19 males in the group of patients with tuberculous otitis media, and 15 females and 51 males in the group of patients with simple chronic purulent otitis media.

Leegaard [32] in a study of 200 cases of purulent otitis media in all age groups, with and without pulmonary tuberculosis, found 20 cases of tuberculous otitis media. Eighty-four of the patients were under 15 years of age.

In this group there were 14 cases of tuberculous otitis media; the greatest number, 7, or 50 per cent, were in the age group between 1 and 5 years. Of the 116 patients over 15 years of age, only 6, all males, had a diagnosis of tuberculous otitis media.

PREDISPOSING FACTORS

Twenty-nine of the cases of tuberculous otitis media followed pulmonary tuberculosis. In the remaining 4 positive cases, 1 patient, a woman, 21 years of age, stated that she had had chronic pulmonary tuberculosis of four years' duration and a discharge from the left ear since childhood. This probably was originally a case of chronic purulent otitis media, with secondary infection with tubercle bacilli. The

TABLE 5.—*Incidence of Chronic Purulent Otitis Media*

Pulmonary Tuberculosis		Head Colds	After Bathing	After Measles	After Scarlatina	After Influenza	Miscellaneous or No Known Cause
Negative cases	18	10	3	2	2	1	30
Positive cases	29*						

* In 4 patients the aural condition was discovered before any notice was taken of the pulmonary condition.

other 3 cases showed recognizable symptoms of chronic pulmonary tuberculosis from five to nine months after the aural condition was noticed. It is possible that there might have been active pulmonary tuberculosis at the time the aural symptoms first appeared.

Eighteen of the nontuberculous cases of chronic purulent otitis media followed chronic pulmonary tuberculosis and apparently had no connection with any other disease. Ten definitely followed colds in the head; 3 were noticed after swimming; 2 followed measles; 2, scarlatina; 1, influenza; 1, an injury; 1, hemoptysis; 1, a "mastoid operation at the age of 5"; another "just came on with bad earache." For 26 no cause could be assigned. Many of these patients had had the aural condition since childhood or it had existed so many years that they had forgotten how it came about. Nearly all these patients had had pulmonary tuberculosis for a relatively short time.

Of the 33 patients with tuberculous otitis media 17 (48 per cent) had other tuberculous lesions outside the chest. Of the 66 with chronic

32. Leegaard, Frithjof: Tuberculosis of the Middle Ear, Laryngoscope **31**:374, 1921.

purulent otitis media 20 (30 per cent) had other tuberculous lesions outside the chest. Eighteen in the entire series had tuberculosis of the larynx; there were about twice as many in the group of positive as in that of negative cases. The other complications included tuberculosis of the meninges, gastro-intestinal tract, pleura and bones and joints.

Aural Involvement.—Among the 33 positive cases both ears were involved in 11, the left ear in 11 and the right in 11.

Among the 66 negative cases there was bilateral involvement in 14, involvement of the left ear in 29 and of the right ear in 23.

ETIOLOGY

The prime etiologic factor is the presence of tuberculosis in the respiratory tract, but apparently it does not necessarily follow that there must be tuberculous involvement of the upper respiratory tract since only 9 of the 33 positive cases showed lesions in the upper respiratory tract. It is significant, however, that among the cases which were diagnosed as simple chronic purulent otitis media there were only 8 showing lesions of the upper respiratory tract, 3 of which were considered clinically positive cases of tuberculous otitis media.

In all the 33 cases of positive tuberculous otitis media except 2 cases of general miliary tuberculosis the sputum was positive for tubercle bacilli.

This sputum, laden with infecting organisms coughed up from the pulmonary tract, becomes trapped in the nasopharynx from which point it reaches the middle ear by way of the eustachian tube. In the majority of cases observed no demonstrable tuberculous lesions were discovered in the nasopharynx, the mucosa of which, as a rule, was congested and thickened. Actual tubercles were observed in the nasopharynx in 2 cases in which tiny yellowish-gray areas the size of the head of a pin were consistently seen in the region of the nasopharyngeal orifices of the eustachian tube. It is my opinion that the size, shape and position of the nasopharyngeal ostium of the eustachian tube have considerable bearing on the facility with which an ear becomes infected with the tubercle bacillus, those with a relatively large, round, more or less protuberant ostium acting more readily as a pathway for tubercle bacilli to the middle ear. Also the inclination of the eustachian tube downward and outward from the nasopharyngeal ostium to the middle ear is undoubtedly a factor. The action of cilia toward the nasopharyngeal ostium tends to keep the tube clear, but, in the presence of a large amount of infected material such as is seen in cases of advanced pulmonary tuberculosis, their activity seems almost futile. A forcible blast of air might also counterbalance their activity. Briggs [28] referred to these factors in 1914. The withdrawal of fat from the body which takes place in a

wasting disease, such as pulmonary tuberculosis, apparently is not an important mechanical factor in tuberculous otitis, because tuberculous otitis may develop late in the course of chronic pulmonary tuberculosis in persons who are gaining weight and apparently overcoming their pulmonary lesions.

The infection may reach the middle ear in a great majority of cases without producing lesions of the eustachian tube itself, when there is easy access for infected material into the tube. In some cases, however, there is evidence to show that the mucosa of the tube itself is infected. Fraser [33] showed by microscopic section the spread of tuberculosis along the eustachian tube. Chapman [20] reported having seen a case of tuberculosis of the nasopharynx which was followed by bilateral tuberculous otitis media.

CHANGES IN THE EAR

Few of the cases coming under observation showed early changes in the tympanic membrane. The earliest change seen was an engorgement of the blood vessels so pronounced that each individual vessel stood out distinctly against the background of the tympanic membrane whose color had changed little from the normal but whose luster was absent. This apparent enlargement of blood vessels became more pronounced before the membrana tympani became definitely red, so that vessels stood out like tiny strands over the surface of the membrane. As the drum membrane became redder and thicker, the cordlike appearance of the blood vessels faded. In one patient in whom, on first examination, the tympanic membrane was red, particularly along the malleus and in Shrapnell's membrane, the process advanced to such a degree that at the end of two weeks the membrane was thickened, deep red and bulging. There was no pain. The temptation to open the ear drum was great, but the operation was not performed, and two months later the inflammation had begun to subside. At no time during the period of observation was there any discharge from the ear.

On the other hand, in a patient under observation because of tuberculous infection of the left ear, a similar infection developed in the right ear and perforated the tympanic membrane painlessly in three days.

Perforations in the ear drums were not multiple in any of the positive or clinically demonstrable cases. In each of the early cases the perforation was so tiny that it could not definitely be seen with the naked eye, but with the magnifying otoscope and careful cleaning of the external canal a small bead of seropurulent material could be seen coming from a perforation in the anterior inferior quadrant of the drum.

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33. Fraser, J. S.: Tubercular Disease of the Ear (Pathology), Proc. Roy. Soc. Med. (Sect. Otol.) **3**:17, 1914-1915.

I have never observed any pulsation such as is seen in an acute pyogenic infection. Careful observations were made for the appearance of tubercles followed by two or more perforations in the tympanic membrane. I never definitely recognized any tubercles and never saw more than one perforation. In some of the cases the perforation did not become large enough to be visible for as long as six weeks.

In other cases there was more or less destruction of the membrana tympani until practically the entire membrane had disappeared. In the cases in which there was marked destruction of the tympanic membrane a large part of the malleus was still visible, and the color of the remaining portion of the membrane was practically normal.

Ormerod [1] observed that multiple perforations and large perforations seem to be the rule and that Shrapnell's membrane is not often involved.

Leegaard [32] stated that multiple perforations of the ear drum are common but that they are frequently seen in nontuberculous otitis also. He also stated that multiple perforations are common in phthisical patients with tuberculous otitis media but not in patients with tuberculous otitis media but free from pulmonary tuberculosis. It is rarely that one actually sees tubercles in the membrana tympani, but the presence of abundant and recurring granulations should arouse suspicions as to the true nature of the disease.

Granulations in the fundus and over the exposed promontory were observed in 5 of the 33 positive and in 3 of the 12 suspected cases of tuberculous otitis.

DIAGNOSIS

Tuberculous otitis media was most frequently seen in young adults with advanced pulmonary tuberculosis, in the greater number of instances less than one year in duration. The first symptom was usually fulness in the ear, noticed after blowing the nose or coughing, followed by deafness which was out of proportion to the apparent involvement of the membrana tympani, not accompanied by pain, and followed by aural discharge after a period of from a few days to a few weeks. Deafness was persistent, rarely showing much if any improvement, even after the ear revealed a tendency to resolution or, indeed, after complete healing had taken place.

Discharge from the ear was usually "watery" and scant for a period averaging two or three weeks after it was first noticed and gradually became purulent and more abundant. However, there were occasional cases in which no discharge was noted but in which on examination a small amount of purulent material was found in the region of the fundus.

The appearance of the membrana tympani was not characteristic in the early stages except that there appeared to be an unusual engorgement of the larger blood vessels over the surface of the membrane

without a marked change in its color. The progressive changes in the drum membrane were infinitely more gradual in sequence than in an ordinary acute purulent otitis media. Usually no pain was noticed during this period, but deafness was pronounced.

It seems that the lesion in the ear showed tendencies similar to those of lesions in the chest; i. e., if the lesions in the chest were exudative, the aural lesion seemed to be of that type, and if there was a tendency to fibrosis in the chest, a similar tendency was noted in the ear.

On the other hand, I have seen a tuberculous otitis media in one ear resolve and become completely healed while the patient's thoracic lesions were becoming more advanced and his general condition more hopeless. I have seen one ear getting well while the other one was being involved and going through all the changes incident to infection that the first one had gone through. Also a case has been seen in which the pulmonary lesions and the general physical condition were showing marked improvement when tuberculous otitis media developed, first in one ear and several months later in the other.

Fleeting twinges of pain were noted by some patients at some time during the course of the disease. Some complained of pain in the external auditory canal, some of indefinite pain over the side of the head on which the affected ear was located and some of pain directly in the ear itself. In all there were 6 cases of tuberculous otitis media in which even the slightest pain was noted, but it was never an annoying symptom or one on which attention was acutely centered. A report of tuberculous otitis media in which pain was the predominating symptom has been made. Harris,[34] in discussing the report of a case of tuberculous otitis by John A. Robinson before the New York Academy of Medicine in 1915, cited a case of advanced pulmonary tuberculosis and tuberculous disease of the middle ear in which there was excruciating pain in the ear extending over days and weeks.

In a majority of Leegaard's [32] cases more or less pain was present. Tinnitus was not constant but was noted at some time during the course of the disease in 16 cases. In 2 cases it was the first symptom complained of, and in 2 others it was constant from the onset until the patient was no longer under observation.

The final diagnosis of tuberculous otitis media can be definitely made only with the aid of the laboratory. Direct smears from the aural discharge are not decisive, nor are they accurate. Wingrave [35] listed six

34. Harris, T. J.: Ann. Otol., Rhin. & Laryng. **24**:363, 1915.

35. Wingrave, Wyatt: The Microscopic Examination of Discharge in One Hundred Cases of Middle Ear Suppuration, with an Analysis of the Results Having Special Reference to the Presence of Tubercle and Acid-Fast Bacilli, Ann. Otol., Rhin. & Laryng. **12**:440, 1903.

acid-fast and alcohol-fast bacilli which may be confused with the tubercle bacillus. Briggs,[28] however, stated a belief that these organisms are encountered so rarely in the ear that they need not be considered important.

Woolley and Petrik[36] used various culture mediums to isolate the tubercle bacillus but decided that it was necessary to seed at least twenty tubes of their best medium to give results equal to inoculation of animals when the animals were given a suitable amount of material.

A partial lack of faith in inoculations of animals has been expressed earlier in this report, but it is believed that the error in this method of isolating the organism is one of omission, and that when positive results are reported there can be no doubt of the nature of the infection.

The laboratory investigation was conducted at Bellevue Hospital by M. S. Lester, whose description of the technic follows:

LABORATORY TECHNIC

The test used is one for the determination of the presence of tubercle bacilli by inoculation of the suspected material into a guinea-pig which has previously shown a negative cutaneous reaction for tuberculosis.

Material is taken from the ear on a cotton swab and placed immediately in a solution of 3 per cent sodium hydroxide to remove contaminating bacteria found in the secretion from the ear in sufficient amount to cause death to the guinea-pig, and also to digest the waxy secretion in which tubercle bacilli may be incorporated. The solution is centrifugated for from twenty to thirty minutes. As sodium hydroxide irritates animal tissue it must be neutralized to phenolphthalein by the addition of a 15 per cent solution of hydrochloric acid. Two and five-tenths cubic centimeters of neutralized material is injected by the subcutaneous method into the groin of a guinea-pig. After two weeks a tuberculin test is made on the animal; 0.1 cc. containing 1 mg. of tuberculin is injected intracutaneously into shaved areas of the abdomen and the reaction noted after forty-eight hours. This test gives an indication of the subsequent outcome of the inoculation, and if the animal has been inoculated with tuberculous material, hastens the eventual outcome. When the animal dies the liver and spleen are examined grossly and microscopically for evidence of tuberculosis. If the animal is still alive at the end of eight weeks, it is killed and gross and microscopic examinations are made.

PROGNOSIS

In none of the cases in this series was death caused by aural tuberculosis. The presence of aural tuberculosis did not even contribute sufficiently to the underlying tuberculous condition to hasten an eventual unfavorable outcome of the disease, nor was there any evidence of the spread of tuberculosis from the aural infection to nearby vital structures.

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36. Woolley, J. S., and Petrik, F. G.: Potato-Egg Medium for Isolation of Tubercle Bacilli, Am. Rev. Tuberc. **24**:596 (Nov.) 1931.

The relatively benign character of aural tuberculosis in adults, as far as its effect on general well-being is concerned, is shown by the following report of cases:

A white man, aged 21, with chronic pulmonary tuberculosis, noticed deafness in his left ear and a discharge of a small amount of pus four months prior to admission to the hospital. Tubercle bacilli were found in the aural discharge. His pulmonary condition progressed rapidly while he was under observation, and the ear drum was almost entirely destroyed, but nine months later the infection in the left ear had resolved, and the ear was healed, although exceedingly deaf. At that time the right ear became involved and was found to be tuberculous. The pulmonary condition apparently could not be controlled, but the aural tuberculosis cleared up entirely in one ear and began to clear in the other before death intervened.

Another patient being treated by pneumothorax for a large cavity in the right lung was constantly improving so far as the pulmonary lesion was concerned, when she became deaf and noticed a discharge from the right ear. This proved to be a tuberculous otitis, and two months later the left ear showed a similar type of infection. The presence of these added infections, however, did not appear to halt the improvement in general condition or to react adversely on the pulmonary lesions.

A case of entirely different type is of interest in this connection.

A man aged 28, born in France, was admitted with a diagnosis of chronic pulmonary tuberculosis of undetermined duration and tuberculosis of the twelfth dorsal and first and second lumbar vertebrae with a cold abscess of one year's duration. His complaints were pain in the back and headache. There was no cough or expectoration, and he had noticed no loss of weight, but he was emaciated, weak and ill. He was very deaf. When he was 4 years of age he had had "many abscessed glands in the neck"; these were opened, drained for a month or so, and healed with unsightly scarring. At the same time the left ear discharged, and a "soft abscess" appeared behind it. This apparently had been opened but no operation on the mastoid had been performed. The left side of the face was paralyzed. When he was 8 years old the left eye was enucleated because of a corneal ulcer. There had been no discharge from the right ear and intermittent discharge from the left, but both ears had shown a pronounced defect of hearing since childhood. On examination pus was seen in both external auditory canals. On the left side there was a great deal of scarring, but Shrapnell's membrane and the short process of the malleus were identified. The landmarks of the right membrana tympani could be seen, but considerable scarring and deformity were seen in the anterior inferior quadrant where there was a perforation of moderate size.

Pus from the cold abscess in the loin was negative for tuberculosis, as were the cerebrospinal fluid and the aural exudate. Ten days after admission to the hospital the patient died of tuberculous meningitis, which apparently was a terminal complication of his other tuberculous lesions. Tubercle bacilli were never found in any of the laboratory examinations made.

In no case under our observation was any surgical intervention considered feasible or necessary. Roentgen examinations of mastoids showed no evidence of breaking-down of cell septums.

No vestibular disturbances were noted in any case.

Up to the present time a relatively small number of cases have been under observation for a long enough period to give a good idea of what finally happens to the ear in aural tuberculosis. A considerable number of patients died of pulmonary tuberculosis. Some showed improvement in the aural condition while the pulmonary disease was distinctly on the down grade, and there were some in whom a definite improvement of the pulmonary tuberculosis was noted at the time tuberculous otitis media developed. As a general rule, however, an improvement in the pulmonary condition and in the patient's general physical condition was accompanied by a similar improvement in the aural tuberculosis. The ability of the majority of these patients to hear was distinctly diminished. There were only 2 patients whose defect of hearing noticeably improved, in one after six weeks and in the other after eighteen months. Twenty-four patients have had persistent deafness for from eight days to three years, without any noticeable subjective or objective improvement.

Few positive cases in this series were observed for a sufficiently long period to determine the average time elapsed before resolution took place. One patient who was under observation intermittently for twenty-three months had no signs of active infection at the end of twenty-one months. Another who was never proved to be tuberculous because there was never any exudate from the ear practically recovered within a period of three months but regained little hearing.

It seems that aural tuberculosis does not materially affect the prognosis of pulmonary tuberculosis, that the tuberculous condition in the ear does not compare in gravity with lesions of the upper respiratory tract, such as tuberculous laryngitis or pharyngitis, but that impairment of the auditory function of the ear is pronounced and permanent.

TREATMENT

Because of inability to keep these patients under observation for long periods, treatment and the results of treatment were not stressed in this study. Various palliative measures were resorted to, such as the instillation of boric acid and alcohol and mercurochrome into the ear. In one case in which alcohol and boric acid had been instilled into the ear for several days, the exudate was found positive for tubercle bacilli a short time after the medication.

This phase of the study will be taken up at another time.

CONCLUSIONS

As a rule, tuberculosis of the middle ear in adults is a chronic disease without appreciable involvement of the temporal bone and not accompanied by fatal complications. It accompanies or follows chronic pulmonary tuberculosis. The sputum is invariably positive for tubercle

bacilli. Four and seven-tenths per cent of patients with chronic pulmonary tuberculosis have otorrhea; 1.7 per cent of these conditions are tuberculous, and the remaining 3 per cent are classed as simple chronic purulent otitis media. Thirty-three per cent of all cases of chronic purulent otitis media occurring in patients with chronic pulmonary tuberculosis are tuberculous.

The statistics of incidence, etiology, pathology, prognosis and treatment are entirely different for children.

The average age of patients with tuberculous otitis media is less than that of patients with simple chronic purulent otitis media.

Tuberculous otitis media is relatively more prevalent among females.

The infection of the middle ear appears to result directly from active pulmonary lesions by way of the nasopharynx and eustachian tube in cases of chronic pulmonary tuberculosis. The infection is evidently hematogenous in the acute cases.

Some significance must be attached to the anatomic relationship of structures in and about the nasopharynx.

Definite tubercles and multiple perforations of the membrana tympani were not observed in this series of cases.

A painless, extremely deaf, chronically discharging ear in a young adult with active pulmonary tuberculosis is usually tuberculous.

The final diagnosis of tuberculous otitis media is made only with the aid of the laboratory.

Tuberculous otitis media is not an alarming complication of chronic pulmonary tuberculosis in adults. However, it produces a marked defect of hearing.

The few nontuberculous patients with otorrhea who were operated on showed no evidence of tuberculous otitis.

SUMMARY

A report has been made which includes a statistical study of the incidence of aural tuberculosis among adults with pulmonary tuberculosis, together with a clinical study of the factors entering into the production of the disease, the clinical course it follows and the effect it has on underlying tuberculous lesions.

INFLUENCE OF FLUORINE ON THE BONY LABYRINTH OF THE WHITE MOUSE

FURTHER OBSERVATIONS

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CHICAGO

A renewed interest has recently been manifest in the biochemical factors bearing on otosclerosis. At the meeting of the German Otolaryngologic Congress in 1932, and previously, at the meeting of the Southwest Society of German Otolaryngologists, Grahe and Griebel [1] reported a series of experiments with the blood serum of known otosclerotic patients on ground up bone of the labyrinth, which gave up its calcium and phosphorus to a greater degree than when exposed to normal serum. They attributed this to the action of a ferment on the order of a phosphatase in the blood of otosclerotic persons.

At the 1933 congress of German otolaryngologists, Krainze [2] reported experiments on rabbits with artificially induced acidosis, resulting in increased porosity of the labyrinthine bone and deposition of uncalcified bone and also general rachitic-like changes in the skeleton.

Barth,[3] using parathyroid extract in rats and guinea-pigs, reported changes in the labyrinth of rats resembling osteitis fibrosa.

Tobeck,[4] by overdosing with viosterol, produced experimentally changes resembling osteodystrophia fibrosa.

Nager,[5] in his article on diseases of the labyrinth, mentioned biochemical experimentation and named some of the workers to whom I refer.

A number of years ago, prompted by the known effects of fluorine on the metabolism of bone, I instituted experiments on feeding white mice with calcium fluoride, and was able to show in some of the mice changes in the labyrinthine bone consisting of a persisting immaturity somewhat similar to the appearance of the ossicles in these animals. These results, with a bibliography and slides, were presented before the American Laryngological, Rhinological and Otological Society in 1928.[6]

Read before the American Otological Society, April 6, 1934.

1. Grahe and Griebel: Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:421, 1932.
2. Krainze: Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:170, 1933.
3. Barth: Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:175, 1933.
4. Tobeck: Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:188, 1933.
5. Nager: Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:72, 1933.
6. Lewy: Influence of Fluorine on the Bony Labyrinth of the White Mouse, Arch. Otolaryng. **8**:315 (Sept.) 1928.

Further study of the specimens reveals, in addition to the changes reported then, the deposition of osteoid tissue on the foot plate of the stapes and on the adjacent labyrinthine wall in one of the mice fed experimentally (figure). The evidence is somewhat clouded by the presence of otitis media, which is common in laboratory animals subjected to experiments in feeding, but nowhere does the inflammatory reaction go on to the destruction of bone, so the inflammation as a cause of the osteoid deposit seems improbable.

As against the pessimistic attitude engendered by the widely accepted hereditary factor in otosclerosis, and by the regression theory of Dr. Guggenheim,[7] who would trace the origin of otosclerosis to genes originating in the ancient ancestry of Proteus anguinas, these experiments,

The effect of fluorine on the bony labyrinth, showing (*x*) osteoid deposits.

together with the evidence of occasional spontaneous arrest, offer hope of the eventual development of therapeutic resources in the management of this unconquered form of deafness.

In conclusion, I should like to mention that since my earlier paper was published, considerable more work has been done on the influence of fluorine on the metabolism of bone. A number of articles have recently been published from the University of Wisconsin laboratories.[8] Smith and Lantz,[9] of the University of Arizona, reported that 0.1 per

7. Guggenheim, L. K.: Tr. Am. Otol. Soc. **23**:75, 1933.

8. (*a*) Phillips: Science **76**:239 (Sept. 9) 1932. (*b*) Lamb, Phillips, Hart and Bohstedt: Am. J. Physiol. **106**:350 (Nov.) 1933. (*c*) Phillips, Lamb, Hart and Bohstedt: ibid. **106**:356 (Nov.) 1933. (*d*) Phillips and Stare: J. Biol. Chem. **107**:351 (Feb.) 1934. (*e*) Halpin and Lamb: Poultry Sc. **11**:1 (Jan.) 1932.

9. Smith and Lantz: J. Biol. Chem. **101**:677, 1931.

cent of sodium fluoride in the ration of rats changes the calcium phosphorus ratio, increasing the calcium level and decreasing the phosphorus. Phillips and Stare [8d] stated that in fluorine toxicosis vitamin C is increased in the actively metabolizing tissues, and especially mentioned the kidneys, liver, anterior lobe of the hypophysis and the suprarenal cortex.

INCIDENCE AND SIGNIFICANCE OF SINUSITIS IN PNEUMONIA

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In a study on the incidence of sinusitis one is impressed not only by the great prevalence of such infection in this locality, but also by the great frequency with which it is found in close association with certain illnesses such as otitis media, mastoiditis, the exanthematous diseases and pneumonia. Statistics on such associations are lacking and, in their absence, it is perhaps not justifiable to attach any great significance to their relationship, but in the instances of otitis media and pneumonia the frequent, almost constant presence of sinusitis at least strongly suggests an etiologic relationship. Two years ago I [1] published the results of a study on the association of otitis media and sinusitis, emphasized the probable etiologic relationship between the two conditions, and advocated energetic treatment of such infections of the sinus, as both a prophylactic and a therapeutic measure in the control of infections of the middle ear.

In this contribution I wish to present my observations on the association of sinusitis and pneumonia. In the past few years, in the examination of several hundred infants and children with pneumonia, I have been impressed by the constant presence of acute sinusitis, but only recently have I attempted to record these observations and to draw any conclusion from them. These observations cover a study of the condition of the sinuses and ears in 130 patients with lobar and bronchopneumonia observed in the Children's Hospital of Philadelphia, the University of Pennsylvania Hospital and St. Christopher's Hospital for Children during the past two years. They represent a consecutive unselected group varying in age from 3 weeks to 90 years. Acute sinusitis was found in 100 per cent of these patients with pneumonia.

METHOD

The procedure of examinations of the nose, throat and ears was as follows: (1) gross inspection of the nose, the degree of congestion of the nasal mucous membrane, the engorgement of the turbinates, the

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From the Department of Otolaryngology of the University of Pennsylvania.

Read before the Section on Otolaryngology of the College of Physicians, Philadelphia, Feb. 21, 1934.

1. Campbell, E. H.: Association of Acute Sinusitis and Acute Otitis Media in Infants and Children, Arch. Otolaryng. **16**:829 (Dec.) 1932.

amount of irregularity of the septum and the amount and character of the secretions being noted; (2) application of a 2 per cent solution of cocaine hydrochloride to the nasal mucous membrane, especially over the lower turbinate and the under surface of the middle turbinate; (3) introduction of the nasopharyngoscope into each nostril, observation being directed especially to the exact location of pus in the nose; (4) examination of the ears, the condition of the drum and the character of the discharge (if any) being noted; (5) examination of the throat with special reference to the presence or absence of postnasal secretions, and (6) roentgen examination of the sinuses in a few selected cases.

The diagnosis of sinusitis was made in practically all the cases by the use of the nasopharyngoscope which I have found more reliable for accurate diagnosis than roentgenograms. In a few instances in which pus could not be seen in the nose, but in which sinusitis was suspected from the intranasal appearance, roentgenograms verified the suspicion.

The use of a 2 per cent solution of cocaine in the nose preliminary to the examination of the infants may be criticized by some physicians, but in hundreds of children, including premature infants, I have never seen the slightest reaction that could be attributed to its use. This has convinced me that the frequently reported reactions that have been attributed to the use of cocaine in the nose have been due, not to the action of the drug (except when used in large amounts), but probably to the psychic disturbances that so often accompany instrumentation in the nose, particularly in persons of the emotional adolescent age.

Of course the amount of cocaine solution used for the younger patients has been very small. A rather tightly wound cotton applicator was dipped into the 2 per cent solution, the excess pressed out and the applicator then rubbed along the floor of the nose, septum, lower turbinate and under surface of the middle turbinate. A minute of rubbing suffices to produce excellent anesthesia and sufficient constriction of the congested nasal vessels and turbinates for satisfactory examination. At times it is necessary to remove some of the purulent or mucous secretion from the nose before a satisfactory view with the nasoscope can be obtained. For this purpose I find the best instrument to be a hand-suction rubber bulb containing a glass tip to fit the nostril. Such an instrument, if used properly, not only removes the secretions lying in the nostrils, but also drains the pus from the sinuses. When the nose is sufficiently clear, the nasoscope is inserted and the location of pus determined. When pus is observed in the middle meatus it may be concluded that it is coming (in infants and young children) from the anterior ethmoid cells, the maxillary sinus or both. When a large amount of pus is present, I have assumed that both sinuses are involved. At times pus will be seen coming from the maxillary orifice in a stream with additional pus around the ethmoidal area, indicating an infection

Cases of Associated Sinusitis and Pneumonia, Showing Variations at Different Ages

Case	Hospital*	Age	Condition of Ears	Examination with Nasoscope	Clinical Examination of Lungs and Diagnosis	Roentgenologic Report	
						Chest	Sinuses
86	C. H.	3 weeks	Bilateral acute purulent otitis media	Large amount of pus in both middle meatuses and spheno-ethmoid recesses	Dulness, bronchial breathing and râles over entire right side of chest; lobar pneumonia; meningitis	Dense shadow over entire right side of chest due to consolidation	
109	St. C.	2 months	Bilateral acute purulent otitis media	4/19/32, moderate amount of pus in both spheno-ethmoid recesses; 5/9/32, large amount of pus in both middle meatuses and spheno-ethmoid recesses	4/18/32, physiologic; 4/23/32, physiologic; 5/1/32, physiologic; 5/6/32, dulness and bronchial breathing over right upper part of chest; broncho-pneumonia; died		
82	C. H.	5 months	Bilateral acute purulent otitis media	Large amount of pus in both spheno-ethmoid recesses; smaller amount in middle meatuses	Dulness and râles over left scapula; pneumonia	Density outward from left hilus; pneumonia, lower part of left lung	
72	C. H.	8 months	Bilateral acute purulent otitis media	Pus in both spheno-ethmoid recesses	Impairment and increased breath sounds over left lung	Small patch of consolidation at base of left lung	
17	C. H.	1 year	Acute purulent otitis media and mastoiditis	No examination made	Prolonged expiration with râles anterior and posterior over right apex; lobar pneumonia	Slight dense shadow over right upper lobe	Both ethmoid sinuses quite dense, antrums less so, thought to be due to pansinusitis
4	C. H.	18 months	Bilateral acute purulent otitis media	Large amount of pus in both middle meatuses and spheno-ethmoid recesses	Dulness and bronchial breathing at right apex posteriorly; lobar pneumonia	Dense shadow in upper half of right lung typical of lobar pneumonia	
79	C. H.	3 years	Bilateral acute purulent otitis media	Small amount of pus in right middle meatus	Dulness, bronchial breathing and râles over lower part of both lungs; chronic broncho-pneumonia	Dense infiltration in lower two thirds of chest on right side	No evidence of pathologic involvement
64	C. H.	6½ years	Normal	Large amount of pus in both middle meatuses and spheno-ethmoid recesses	Impairment and increased breath sounds in right axillary region; less degree over lower lobe, râles, lobar pneumonia		
63	C. H.	9 years	Normal	Large amount of pus in both middle meatuses and spheno-ethmoid recesses	Impairment and bronchial breathing in lower part of both lobes, lobar pneumonia		
57	C. H.	11 years	Normal	Small amount of mucopus in both middle meatuses	Flatness over entire right side of the chest except at apex; râles; lobar pneumonia		
65	U. P.	56 years	Normal	Small amount of pus in left middle meatus and spheno-ethmoid recess	Impairment of right apex to angle of scapula; bronchial breathing and râles; lobar pneumonia	Acute pneumonic process in right lower and middle lobes	

70	U. P.	62 years	Left acute purulent otitis media	None made	Dulness and crackling râles in right axilla	Lesion at apex of right lower lobe; appearance of consolidation	Clouding of left maxillary and both frontal and ethmoid sinuses
68	U. P.	90 years	?	Polypi and pus in left middle meatus; chronic maxillary and probably ethmoid sinusitis	Dulness with fine and coarse râles over right middle lobe; bronchopneumonia	Lesion of part of right lower lobe; appearance of pneumonia	
84	C. H.	9 months	Bilateral acute purulent otitis media	Mucopus in both nostrils; source indefinite	Impairment and râles over left side of chest; lobar pneumonia	Deficient aeration in lower left part of chest	Increased density in both antrums and ethmoid sinuses due to infection; suggestion of pus
87	C. H.	3 months	Bilateral acute purulent otitis media	1/24/32, large amount of pus in both middle meatuses and spheno-ethmoid recesses	1/20/32, scattered râles in both lungs; chest resonant; 1/31/32, impairment over left base; bronchovascular breathing over base of right lung; bronchopneumonia	1/21/33, density in left lung behind cardiac shadow	1/25/33, antrums appeared too small to be source of infection
75	C. H.	2 years	?	No acutal pus seen, but swelling and congestion of mucous membrane seen in spheno-ethmoid recesses	Impairment, tubular breathing and fine râles over right upper lobe; lobar pneumonia	Dense shadow over two thirds of right upper lobe	Antrums clear; increased density in both ethmoid areas, especially in posterior cells
91	C. H.	16 months	Bilateral acute purulent otitis media	Large amount of mucopus in both spheno-ethmoid recesses	Dulness and râles below right scapula; bronchopneumonia	Dense area in lower third of right upper lobe and middle lobe	Ethmoid sinuses and antrums dense due to infection; suggestion of pus
92	C. H.	2½ years	Bilateral acute purulent otitis media	2/1/32, no pus seen in right nostril; nasoscope could not be inserted in the left	1/15/32, clear throughout; 1/22/32, impairment and râles over right upper lobe; lobar pneumonia	1/18/32, dense area in right upper lobe due to pneumonia	1/18/32, density in left antrum and ethmoid due to infection; suggestion of pus
93	C. H.	3 months	Bilateral acute purulent otitis media	Large amount of mucopus in both middle meatuses	Dulness, increased breath sounds over upper part of right lung; bronchopneumonia; death	Patches of increased density in both lungs due to pneumonia	Impossible to distinguish between antral infection and lack of development
14	C. H.	2 years	Bilateral acute purulent otitis media	No pus seen in middle meatuses or spheno-ethmoid recesses	Dulness and bronchial breathing over upper part of right lung; lobar pneumonia	Dense shadow in entire right upper lobe due to pneumonia	Both antrums and ethmoid sinuses very dense owing to pansinusitis with retention of pus
16	C. H.	8 months	Normal	No pus seen coming from any of the sinuses on two examinations	Dulness, diminished breath sounds and râles over entire upper lobe of left lung; lobar pneumonia	Dense shadow in left upper lobe; due to lobar pneumonia	Both antrums and ethmoid sinuses small and very dense, thought to be due to infection with retention of pus
18	C. H.	11 months	Bilateral acute purulent otitis media	No examination made	Impairment and suppressed breath sounds over upper lobe of right lung; lobar pneumonia	Dense shadow in entire right upper lobe; typical of lobar pneumonia	Both antrums and ethmoids very dense - owing to pansinusitis with retention of pus

* C. H. indicates the Children's Hospital of Philadelphia; U. P., the University of Pennsylvania Hospital, and St. C., St. Christopher's Hospital for Children.

Cases of Associated Sinusitis and Pneumonia, Showing Variations at Different Ages—Continued

Case	Hospital*	Age	Condition of Ears	Examination with Nasoscope	Clinical Examination of Lungs and Diagnosis	Roentgenologic Report	
						Chest	Sinuses
13	C. H.	11 months	Bilateral acute purulent otitis media	4/5/33, large amount of pus in both middle meatuses and spheno-ethmoid recesses	4/4/33, physiologic; 4/12/33, dulness and bronchial breathing over base of left lung; lobar pneumonia	Diffuse shadow in base of left lung; thought to be consolidation of a pneumonia	
11	C. H.	7 years	Normal	4/4/33, pus in both spheno-ethmoid recesses	4/3/33, physiologic; 4/6/33, dulness, bronchial breathing and râles over upper lobe and base of left lung; lobar pneumonia	Definite area at base of left lung, due to patch of pneumonia	
1	C. H.	14 months	Bilateral acute purulent otitis media	1/11/33, pus in both middle meatuses and spheno-ethmoid recesses	1/4/33, physiologic; 1/15/33, percussion note impaired, and bronchial breathing over upper lobe of right lung; lobar pneumonia	1/15/33, dense area over upper right lobe, typical of lobar pneumonia	
4	C. H.	18 months	Bilateral acute purulent otitis media	3/3/33, large amount of pus in both middle meatuses and spheno-ethmoid recesses	2/25/33, physiologic; 2/28/33, dulness and bronchial breathing at apex of right lung posteriorly; lobar pneumonia	Diffuse shadow in upper half of right lung typical of lobar pneumonia	
101	St. C.	2½ months	Right acute purulent otitis media	2/8/32, large amount of pus in right middle meatus and spheno-ethmoid recess	2/1/32, physiologic; died 2/29/32 with bronchopneumonia		
105	St. C.	4 months	Bilateral acute purulent otitis media	2/15/32, small amount of pus in both spheno-ethmoid recesses	2/8/32, physiologic; 2/27/32, physiologic; 3/9/32, dulness; harsh breathing sounds at apex of left lung; râles on both sides of chest; bilateral bronchopneumonia; Little's disease		
109	St. C.	2 months	Bilateral acute purulent otitis media	4/19/32, moderate amount of pus in both spheno-ethmoid recesses; 5/9/32, large amount of pus in both middle meatuses and spheno-ethmoid recesses	4/18/32, physiologic; 5/1/32, physiologic; 5/6/32, dulness and bronchial breathing over right upper part of chest; bronchopneumonia		
111	St. C.	9 months	Bilateral acute purulent otitis media	2/6/32, mucopus in both middle meatuses	1/25/32, physiologic; 1/30/32, physiologic; 2/19/32, scattered areas of impairment and râles over entire chest; bronchopneumonia		
102	St. C.	2 months	Bilateral acute purulent otitis media	3/28/32, large amount of pus in both middle meatuses and spheno-ethmoid recesses	2/27/32, physiologic; 4/2/32, cough and fine râles in chest; 4/3/32, died, bronchopneumonia		
73	U. P.	20 months	Bilateral acute purulent otitis media	2/6/34, large amount of pus in both spheno-ethmoid recesses and left meatus	2/5/34, physiologic; 2/8/34, impairment; harsh breathing sounds and râles over right lower part of chest; lobar pneumonia	2/8/34, acute process of pneumonia of right lower lobe	

* C. H. indicates the Children's Hospital of Philadelphia; U. P., the University of Pennsylvania Hospital, and St. C., St. Christopher's Hospital for Children.

in both sinuses. When the pus is seen in the spheno-ethmoid recess it indicates chiefly infection in the posterior ethmoid cells, but also at times infection of the sphenoid especially in older children.

In this series of 130 patients with pneumonia 52 were under 1 year of age; 24 between 1 and 2 years; 47 from 2 to 15 years, and 7 were adults. It is unfortunate that more adult patients were not examined, but I attempted only recently to include adults in this series and only 7 have been available for my study. In all but 10 of the patients a positive diagnosis of sinusitis was made by the nasoscope. Five of these 10 patients were not examined with the nasoscope, and in each of these the roentgenologic report indicated infection in the sinuses. In only 5 patients was there failure to observe actual pus coming from the sinuses. Two of these showed such a congestion and hyperplasia of the mucous membrane in the middle meatuses or spheno-ethmoid recesses that there was little doubt as to the diagnosis. All five gave roentgenologic evidence of infected sinuses. A positive diagnosis of sinusitis was therefore made in every case. Roentgen examination of the sinuses was made in 15 patients of this series. In one of these the report stated there was no evidence of pathologic involvement. In this patient the nasoscope showed definitely a small amount of pus in the right middle meatus. In another patient aged 3 months the report from the roentgenologist was that the antrums were so small that he thought they could not serve as a source of infection. In this patient a large amount of pus could be seen with the nasoscope in both middle meatuses and spheno-ethmoid recesses. Regarding another patient aged 3 months the roentgenologist stated that it was impossible to distinguish between an antral infection and lack of development. This patient showed a large amount of pus in both middle meatuses. I have found that the diagnosis of sinus infection in infants and children can be more accurately made by the nasoscope than by roentgenograms. In the great majority of instances a single examination with this instrument is all that is needed, but in a few instances two or more examinations may be necessary before a discharge of pus from a sinus is detected.

In 59 of the patients pus was found in the middle meatuses and spheno-ethmoid recesses of both sides; in 31 patients, in both recesses only; in 7 patients, in the middle meatus and spheno-ethmoid recess of only one side; in 6 patients, in one spheno-ethmoid recess; in 10 patients, in both middle meatuses only; and in 2 patients, in one middle meatus only. Of the patients whose sinuses were examined with roentgen rays 9 were found to have increased density in both antrums and ethmoids; one, density in the left antrum and ethmoid, and one, density in both ethmoids.

These statistics show that in 45.4 per cent of the cases there is probable involvement of all the sinuses; in 23.8 per cent, probable involvement

of the ethmoid sinuses alone, and in 5.4 per cent, probable involvement of all the sinuses of one side. Further study of these records shows that involvement of all the sinuses usually occurs among the youngest of the children. Of the 52 children under 1 year of age 27 had probable involvement of all the sinuses; 18 had probable involvement of the ethmoid sinuses only, and the remainder had involvement of one or more of the sinuses. This indicates what has been previously observed by me and by others that in younger children involvement of the ethmoid sinuses is more common than involvement of the maxillary sinuses, but in infants involvement of all the sinuses is likely to occur.

Among the patients examined 8 were of special interest from the standpoint of possible etiologic relationship between the sinusitis and pneumonia. Records of these patients show that examination of their noses revealed pus discharging from the sinuses at the time when the lungs were entirely normal. The records show that within a few days pneumonia developed in these patients. This observation, of course, does not prove that the pneumonia developed from the sinusitis, but only that the sinusitis was present before the pneumonia occurred and was a possible source of the subsequent pulmonary infection. When the great prevalence of sinusitis is considered it appears that pneumonia develops in only a small percentage of such cases. That percentage is sufficiently high, however, to warrant consideration of such infections of the sinus as possible sources of pneumonia and to demand energetic treatment of such sources as a prophylactic measure.

In the 130 patients clinical examination showed that pneumonia was lobar in 82 and bronchial in 48. Empyema developed in five of the patients with lobar pneumonia. The diagnosis of pneumonia was confirmed by roentgen examination in 71 cases. No apparent connection between the type of pneumonia and the amount of sinus involvement was observed except that on the whole the pneumonia appeared to be more of the bronchial type when there was a large amount of discharge from the sinuses. However, as the bronchopneumonias were found more often in the younger children who were unable to remove their nasal secretions, this appears to be of little significance.

An interesting feature in these patients with pneumonia was the observation of the condition of the ears. Acute purulent otitis media developed in a surprisingly large percentage preceding or during the pneumonia. My records show that there was a bilateral acute purulent otitis media in 79 patients, or 60.7 per cent, a one-sided acute purulent otitis media in 12, or 9.2 per cent, an acute catarrhal otitis media in 14, or 10 per cent, and no involvement of the ears in 18, or 13.8 per cent; in 5 cases the ears were not examined. Twelve of these patients were operated on for mastoiditis. In some of those listed under catarrhal otitis or normal ears without doubt a purulent otitis developed

later; I did not follow the aural condition to the end of their illness. Of the 18 patients in whom the ears were normal only 1 was under 1 year of age. The rest were from 3 to 14 years of age and adults. These statistics bear out my observation recorded two years ago, that the incidence of acute otitis media in sinusitis is much greater in younger children and infants, and that the younger the child, the more apt he is to have a purulent otitis media accompanying his sinus infection.

What significance might such a constant association of sinusitis and pneumonia have? It seems that there may be an important one from the etiologic standpoint. It is well known that nasal secretions are frequently inhaled to a greater or less extent, and if infected material from the sinuses is inhaled, direct transmission to the lungs is easily possible. This appears to be true especially in the young children and infants who are incapable of removing their nasal secretions, and it is among this class of patients that one sees the great prevalence of pneumonia. As the child becomes older there is a progressively lower incidence of pneumonia with a progressively lower incidence of sinusitis. I believe that the ability of the older child to remove the excess nasal secretions to some extent has much to do with this lower incidence of pneumonia in sinusitis. In the younger children who are not able to remove the infective nasal secretions, it seems advisable to institute some procedure of removing it for them as far as is practical. The nasal suction bulb seems well adapted to this purpose and I have had such bulbs placed in the wards of two children's hospitals for use on patients in whom sinusitis is discovered. I believe that the frequent and efficient use of such an instrument early in the sinusitis of infancy would greatly lessen the liability to such complications as otitis media and pneumonia.

I do not wish to emphasize too strongly an etiologic connection between the sinusitis and pneumonia. My object is to point out the apparently constant presence of sinusitis in patients with penumonia and the possibility of an etiologic significance in their association.

SUMMARY

1. A study of 130 patients with pneumonia between the ages of 3 weeks and 90 years showed the presence of sinusitis in 100 per cent.

2. The diagnosis of sinusitis was made in all these patients except 10 by means of the nasopharyngoscope. In the remaining 10 the diagnosis was made by roentgenograms.

3. A high incidence of acute purulent otitis media (70 per cent) was found in these 130 patients.

4. It is suggested that infected sinuses are a possible etiologic factor in the production of pneumonia and that the early efficient removal of the purulent secretions from the sinus by suction might tend to lessen the incidence of pneumonia in infants and children.

1904 Spruce Street.

Clinical Notes

RHINOLITH

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The formation of a rhinolith of enormous size in the nose is of curious interest. One must observe and remove it to appreciate its uncanny development in the nasal cavity, and to realize to what stupendous size it may progress before the patient seeks relief for nasal obstruction with its associated fetid odor, discharge and other discomforts.

The largest rhinolith recorded in the literature weighed 110 Gm., had destroyed the septum and turbinate and required Rouge's external operation for its removal.[1] Melay[2] described a nasal calculus which weighed 85 Gm. and caused symptoms suggesting a malignant growth. Myerson[3] reported a nasal calculus weighing 56 Gm., which was so tightly wedged between the septum and lateral wall that he was obliged to remove it through an external exposure of the nasal chamber. Kelemen[4] performed a Denker operation to take out a large concretion which had necrosed the lateral wall and filled the antrum. Gorman[5] used a general anesthesia to extract a calculus that weighed 70 Gm. Cases of rhinolith were reported by Hadley[6] (weight, 48 Gm.), Bailey[7] (weight, 49 Gm.), Scott[6] (weight, 43 Gm.), Bovin of India[6] (weight, 11 Gm.), and two Spaniards, Horcasitas and Calatrabino[6] (each reported gigantic nasal calculi but did not give the weights).

REPORT OF A CASE

K. T., a white woman, aged 45, appeared in the outpatient department with a history that she had always been in good health but that the left nostril had been blocked for two years. A short while before presentation for treatment she had noticed a foul odor, occasional bleeding from the left nostril and pain and buzzing in the left ear. The right middle turbinate was hyperplastic. The left nasal cavity was filled with a dark, hard substance covered with foul secretions. Posterior rhinoscopy revealed the mass extending backward to cover the orifice of the eustachian tube (fig. 1). The right ear was normal. The left ear drum had a moderate amount of retraction with whispered voice heard at 8 feet (2.44 meters).

Operation was performed under local anesthesia; the greater portion of the rhinolith was removed piecemeal with a Knight forceps. The posterior part of the concretion was pushed back into the nasopharynx and removed through the mouth. On account of a moderate amount of bleeding and pain the operation was com-

From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, and the Research and Educational Hospital.

1. Motey, R.: Ann. d. mal. de l'oreille du larynx **32**:496 (Nov.) 1906.
2. Melay, A. B.: Ann. d. mal. de l'oreille, du larynx **32**:91 (Jan.) 1906.
3. Myerson, M. C.: Laryngoscope **38**:393 (June) 1928.
4. Kelemen, G.: Ztschr. f. Laryng., Rhin. & Otol. **15**:93 (Nov.) 1926.
5. Gorman, J. R.: Tr. Am. Laryng., Rhin. & Otol. Soc. **36**:498, 1930.
6. Quoted by Gorman.[5]
7. Bailey, F. W.: Ann. Otol., Rhin. & Laryng. **34**:814 (Sept.) 1925.

pleted in two sittings. The turbinates on the left side were found flattened and pale from pressure. The rhinolith weighed 10.4 Gm. (fig. 2). The photograph visualizes most of the mass pieced together on a block. A nasal spray of menthol and liquid petrolatum was given for home use. Examination made four days later showed the left nasal cavity to be clean and roomy and the left ear drum less retracted and less painful; the fulness had disappeared, and whispered voice was heard at 12 feet (3.6 meters). On transillumination the left ethmoid and antrum appeared cloudy. Satisfied with the complete relief of the nasal and aural condition, the patient objected to further study of the sinuses and ears and of her general condition for a metabolic disturbance as a possible basis for the nasal condition.

Rhinoliths occurring in the gallbladder, kidneys, salivary glands or any secretory gland are of endogenous origin; those occurring in the nose, with a foreign body, necrosed tissue or an infection as a nucleus, are of exogenous origin. The rhinolith

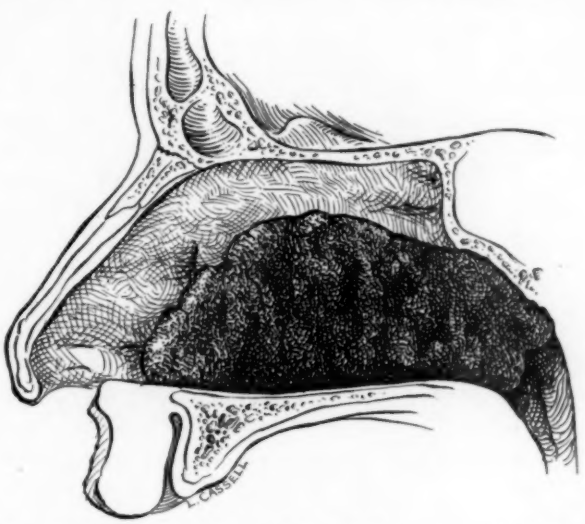

Fig. 1.—Drawing illustrating the position of the mass against the lateral wall and its extent posteriorly to cover the eustachian orifice.

develops from an accumulated deposition of the phosphates and calcium salts from the nasal mucus and tears. These salts are found combined with sodium chloride, calcium carbonate, magnesium and soda, mucin and protein. The formation of a rhinolith is supposedly favored by a gouty diathesis or by some alteration in the character of the nasal secretions that causes the basic salts to become deposited. There is no definite established foundation for this suggested etiologic factor. The rhinolith may be only part of the pathologic condition, and it may occur with any nasal disease associated with excessive secretion. The concretions which contain no definite nucleus form round a nucleus of blood and mucus.

Large calcareous deposits in the nose are not commonly found. The rarity of their appearance is due to the fact that foreign bodies are readily extruded from the nose by blowing or sneezing. In a large clinic Joukovsky[8] observed four

8. Joukovsky, A.: Ann. d'oto-laryng. (Feb.) 1931, p. 147.

cases in twenty-five years. Thomson [9] stated that about three hundred cases have been recorded. Bailey,[7] who reported one case in twenty years, made a survey of the literature and found only forty-nine cases published in the fifteen years preceding 1925. From 1925 to about 1933, a period of approximately seven years, fourteen cases of rhinoliths, including the four of Joukovsky, are found listed in the *Quarterly Cumulative Index Medicus.*

Nasal calculi vary in size from that of granules to gigantic masses completely obstructing the nasal chamber. They are dark brown or coal black and usually hard and brittle. They become covered with granulations and an offensive mucopurulent material which is caused by pressure atrophy of the adjacent tissue and subsequent decomposition. Rhinoliths occur in one side of the nose only. No bilateral or multiple concretions have been observed; neither has a case with recurrence been noted.

The diagnosis of rhinolith offers no difficulty with a proper intranasal examination. A complete diagnosis may not be possible until the concretion is removed.

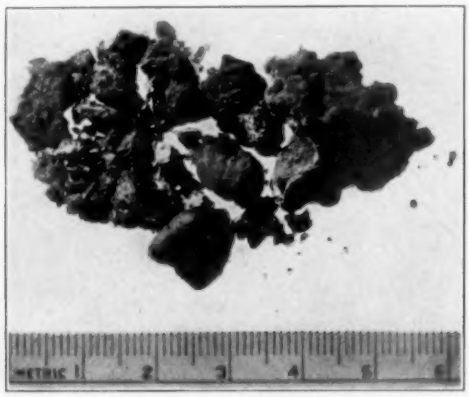

Fig. 2.—The rhinolith has been removed; most of the fragments are pieced together.

All of the cases reported have been in adults, mostly women ranging in age from 28 to 70; in a collection of one hundred cases Seeligman [10] found that sixty-two were in women and twenty-nine in men, the sex not being recorded in nine cases. The history indicates a unilateral nasal obstruction usually of several years' standing and, in the later stages, a foul odor and discharge with dull pain in the cheek or ear, vestibulitis from the irritating discharge and occasional epistaxis on the involved side. These concretions appear to occur most commonly in the clinical patient, which accounts for the long lapse of time permitted before relief is sought. Intranasal inspection reveals a dark mass covered with granulations and foul-smelling secretions filling one nasal cavity. On manipulation with a probe and heavy forceps the mass is found to be hard, immovable and brittle. In the differ-

9. Thomson, St. Clair: Diseases of the Nose and Throat Comprising Affections of the Trachea and Oesophagus; a Textbook for Students and Practitioners, London, Cassell & Co., Ltd., 1926.

10. Seeligman: J. Laryng. **7**:133, 1893.

ential diagnosis atrophic rhinitis, foreign body, syphilitic sequestration, calcified polyps and malignancy are to be considered, but a mere casual analysis should not confuse any of these conditions with a rhinolith.

The treatment is, of course, removal. In the majority of cases local anesthesia will suffice, while general anesthesia is reserved for the occasional noncooperative or painful case, such as that reported by Gorman, and for cases in children when operative trauma is to be avoided. The foremost section of the brittle mass can be removed piecemeal with a Knight forceps or a Killian septum forceps. The posterior portion of the concretion is then pushed into the pharynx and obtained through the mouth. When one is confronted with an unusually large, tightly wedged calculus with marked destruction of tissue, as in the cases cited by Myerson and Kelemen, an external approach under general anesthesia will be required. After extraction of the calculus the turbinates will be found greatly atrophied from pressure. Ulceration may be present. For postoperative care the usual nasal hygiene is observed, and a bland oil spray is freely used.

SUMMARY

1. A large calculus in the nose is rather rare and forms the basis of this report.

2. It results from a continued accumulation of the phosphates and calcium salts deposited from the nasal mucus and tears, with some foreign matter, blood or mucus as a nucleus.

3. Its formation is supposedly favored by some metabolic disorder.

4. Rhinoliths vary in size from a small granule to enormous masses completely obstructing the nasal chamber.

5. They occur in adults, mostly females, and only in one side of the nose.

6. They never recur after removal.

7. Extremely large rhinoliths may require radical operations for their removal.

8. Large concretions may contribute to sinus disease and disease of the middle ear.

55 East Washington Street.

A SET OF INSTRUMENTS FOR ELECTROSURGICAL TONSILLECTOMY

GEORGE B. JOBSON, M.D., FRANKLIN, PA.

I have designed a set of instruments for the electrosurgical removal of tonsils which I believe to be the method par excellence in adults preferably under local anesthesia. If general anesthesia is selected, I choose avertin as being more safe than ether, owing to the danger from explosion of the latter. The instruments [1] used are: an insulated snare, furnished with no. 7 wire, which gives an unob-

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fifth Annual Meeting of the American Medical Association, Cleveland, June 14, 1934.

1. These instruments are made by V. Mueller & Co., Chicago.

structed view of the field and allows easy manipulation; a pair of tonsil forceps, which grasp the tonsil firmly and will not slip; a spud-shaped dissector for freeing the mucous membrane from the tonsil, and an insulated tongue depressor which

Fig. 1.—Insulated snare for electrosurgical tonsillectomy.

Fig. 2.—Tonsil forceps.

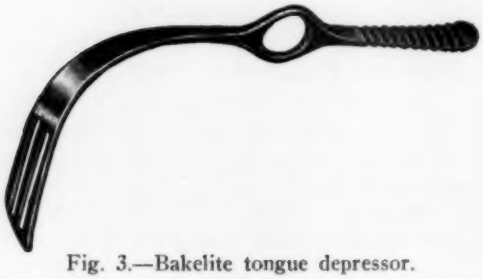

Fig. 3.—Bakelite tongue depressor.

is a modification of the Pynchon instrument. The handle of the depressor is sufficiently long that when grasped the hand of the assistant will not interfere with the operator. An endothermy machine furnishes the cutting current. Under local anesthesia, I have the patient sit on a wooden chair, with the inactive electrode in

contact with the bare buttocks. If the patient is lying on a metal table, the top should be insulated with rubber mats.

The tonsil is seized with the forceps and drawn toward the median line. With the dissector the mucous membrane is freed from the tonsil, to and beneath the margin of each pillar and at the junction of the pillars with the palate. The loop of the snare wire, which is the active electrode, is applied over the forceps, which remain attached to the tonsil, and is then carried to, and placed beneath, the inferior pole of the tonsil, with the tip of the snare in the supratonsillar fossa. The tonsil is drawn toward the midline, the snare compressed and the current turned on with a foot-switch; the tonsil comes away, leaving a dry fossa.

The advantages are: practically a bloodless operation, slight edema, less post-operative pain and less scarring than by other methods.

In three hundred and fifty-eight cases there was slight secondary hemorrhage in three, which was controlled by styptics. In one case the hemorrhage occurred ten hours after the operation and in the other two, three and five days later, respectively.

REMOVAL OF THE NASAL SEPTUM

CARROLL SMITH, M.D., SPOKANE, WASH.

A man, aged 48, complained of a nasal obstruction on the right side of many years' duration, which he believed resulted from a fractured skull. He also complained of dripping from the right nostril. On examination I thought the right nostril was filled with pale polypi, but I found only thick mucus. There was some polypoid tissue in the anterior ethmoid region. The antrum was clear on transillumination, and on irrigation was found to be free of pus.

The septum was straight, but the right posterior naris was absolutely closed. The right eustachian tube could be inflated easily through the left nostril. The nasopharynx was normal. The atresia was due to a continuation of the bony septum at an absolute right angle. This, of course, had nothing to do with the injury to the skull, but was, I believe, congenital.

Because of reports in the literature of the difficulty of keeping a permanent opening in atresia of the posterior naris and because of only fairly satisfactory results in a few cases that I had had, I decided to remove the entire septum. I removed all the septum except the anterior cartilaginous portion and the right-angled posterior portion, removing also the polypi and anterior ethmoid bones. The result was entirely satisfactory.

I could find no report of such an operation in the literature.

Several months previously I had done submucous resections on two adults, with unsatisfactory results as regards the improvement of breathing space. Both had narrow noses and thick septums, and although the septums were straight, mouth breathing was necessary. In one case I tried several times to thin the septum by removing a portion of it, leaving as much of the mucous membrane as possible. The result was still not satisfactory. I finally removed the septum in each patient, both proving to be 9 mm. thick. The results were satisfactory. I have seen the

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Read before the western section of the American Laryngological, Rhinological and Otological Society, Portland, Ore., Jan. 13, 1934.

patients several times since the operation and both say they are sorry that the operation was not done years ago. There was no crusting in the three cases.

I have also performed this operation in two cases of chronic anterior ethmoiditis with polypi that recurred. In both these cases the nose was very narrow and the septum very thick, especially in the upper anterior portion. The frontal sinuses occasionally became acutely involved. After removing the upper anterior portion of the septum it was easy to work through the nostril on the opposite side to clean the area thoroughly. It was also easier to curet and enlarge the frontal sinus opening in these cases through the opposite nostril.

Recently I found an article by McGivern,[1] who believes that "many times the submucous resection should be dispensed with and a complete septal resection done." He reported doing it three times in patients with hay fever and three times in patients with hyperesthetic rhinitis on the advice of the late Dr. Ross Skillern, who he said had done this operation with satisfactory results for years. The sneezing that was bothersome in these cases was completely relieved, and crusting did not result. He referred to the "Fein operation."

In a recent personal communication, however, McGivern said that he has substituted for the complete removal of the septum the removal of the cartilage, bone and mucous membrane from one side, cauterizing the remaining mucous membrane vertically and sagitally, which, he claimed, "shrinks the septum to its proper proportions."

The Fein[2] operation, to which McGivern referred, antedated submucous resection of the nasal septum, and consisted in the removal of the deflected or obstructing portion of the septum.

I believe there are distinct indications for the removal of the nasal septum and that it is done too infrequently. It is not uncommon to hear the remark that submucous resection of the nasal septum is not a satisfactory operation. No doubt some of the resections have not been well done, but some have, and because of the thickened septum the result is disappointing.

As advised by Skillern, indications for the operation, aside from those of hyperesthesia and hay fever in a very narrow nose, are: first, in certain cases of atresia of a posterior naris; second, in certain cases of anterior ethmoiditis with polypi that recur, accompanied by marked thickening of the septum in the upper anterior area, and, third, in patients with a very thick hyperplastic septum in a narrow nose, especially when the septum has not contracted in a reasonable time after a good submucous resection has been performed.

It is wise to study the cases carefully before deciding on this operation. A patient with a very thick septum, especially if the hypertrophy is on one side, should have a Wassermann test, for tertiary lesions of similar appearance disappear promptly with proper treatment.

The operation is not difficult. If a submucous resection has previously been done, the septum can be loosened all around with a knife, and nearly all of it can be removed in one piece; a biting forceps can easily remove the remainder. In two of my patients I checked the bleeding points with the electric cautery so that packing was not necessary.

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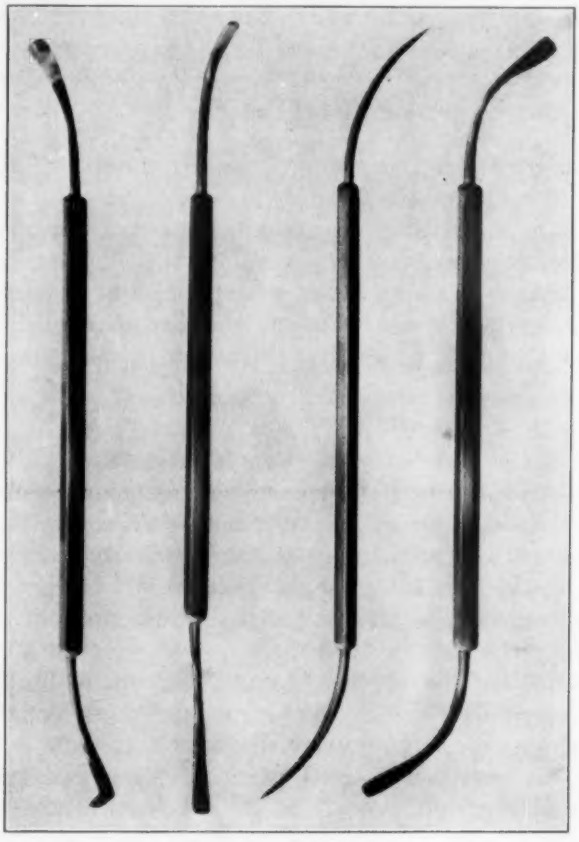
1. McGivern: Suggestions for Modification of Operative Procedure and Postoperative Treatment of Submucous Resections, J. M. Soc. New Jersey (Jan.) 1930.

2. Fein: Ein Beitrag zur Vereinfachung der Submukosen Fensterresektion, Monatschr. f. Ohrenh. **43**:561, 1909.

ADJUSTABLE NASAL ELEVATOR

O. H. Homme, M.D., Los Angeles

This new type of mucous membrane elevator for radical operation on the sinus or for submucous resection measures 9¼ inches (23.5 cm.) in length. The aluminum shaft is hexagonal and of ample dimensions so that it may be held in



Adjustable nasal elevator.

the fingers without cramping the hand. The ends are made of silver, which can be shaped to suit individual requirements. If the silver becomes too hard, as it will in time, it may be made more flexible by being heated in a flame and allowed to cool gradually. The silver is rigid enough to be used in submucous resections or for removing the mucous membrane in operations on the sinus.

With an instrument of this type it is possible to peel the mucous membrane from the sinus walls, and a curet will not be necessary unless there is osteitis.

The instrument was made by the Surgical Mechanical Research Corporation, 531 South Witmer Avenue, Los Angeles.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology.

ALLERGY AS RELATED TO OTOLARYNGOLOGY

W. W. DUKE, M.D.
KANSAS CITY, MO.

The year's literature on allergy is largely a technical development along lines which have been taken up in former years in these reviews. Nothing has appeared which is radically different from material presented in former years, excepting possibly on the subject of so-called ionization. This is in reality an old subject which has been revived and modified in ways which seem to me to be rather unessential and a trifle commercial. However, present-day discussion of the subject warrants comment.

ETIOLOGIC STUDIES

Kerr, Pascher and Sulzberger[1] report that fungi of the Monilia group are found in nearly all skins as well as in the toe-nails, mouth and gastro-intestinal tract of normal persons. They state that fungi of this group are strong sensitizers and are capable of producing primary infections of the skin and secondary eczematous eruptions. In this respect the fungi of the Monilia group resemble those of the Trichophyton group.

The authors find that sensitiveness to either type of fungi is specific for each group; i. e., the groups are not the same immunologically. One patient sensitive to both organisms reacted to each, but in a way which was characteristic of each organism. Also, reactions to intracutaneous and patch tests demonstrated a difference between the two groups of organisms. Treatment by desensitization was found to be specific for the fungus used in desensitization.

Clinical improvement followed specific therapy in some cases, but failed in others. It was considered possible, but not proved, that secondary invaders in certain cases were factors which made the difference between success and failure.

The authors recommend combined treatment with both types of fungi in eczematous eruptions of the hands and feet due to fungi.

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1. Kerr, Phyllis S.; Pascher, Frances, and Sulzberger, Marion B.: Monilia and Trichophyton Extracts: Their Combined Use in Eczematous Ringworm (Dermatophytosis and Dermatophytids), J. Allergy **5**:288, 1934.

Swineford and Weinberg [2] report a case of sensitiveness to heat in which attacks of asthma could be precipitated by as little heat as that encountered by exposure of the chest to an ordinary 40 watt lamp at a distance of 1½ feet (45.7 cm.) for a period of thirty seconds. Attacks brought on this way could be relieved in three or four minutes or less by cold. The patient was successfully desensitized to heat by foreign protein shock obtained through the use of typhoid vaccine.

Duke [3] reports on a study of soy bean as a possible important cause of nasal and bronchial symptoms in persons exposed to dust from soy bean products and foods. Five persons who were subject to cough and asthma in a soy bean mill gave positive reactions to cutaneous tests with an extract of soy bean. Only one person could be studied in detail. He gave intensely positive cutaneous reactions to soy bean extract. This sensitiveness could be transferred by the Prausnitz-Küstner method. Immediate relief was obtained by avoidance of soy bean dust, and partial relief was obtained through specific therapy with soy bean extract. The patient thereafter remained free from asthma, even when exposed to small amounts of the dust.

Soy bean has a wide commercial use. It is used in breakfast foods, diabetic foods, cakes, muffins, biscuits, infant foods, crackers and macaroni. It is also used in soups, coffee substitutes, fresh and condensed milk, and in confections and sauces. It is also used as a canned vegetable. The waste products are commonly used for fodder and fertilizer. Soy bean oil is also used as a substitute for butter, lard and salad oil. It is almost impossible, therefore, for a person sensitive to soy bean to avoid contact with it completely. This protein can be used to advantage in specific desensitization, when a patient's illness can be definitely proved to be due to contact with soy bean products.

Osgood [4] finds that in a case of sensitiveness to Caddis fly the reagin content of the serum proves that one type of Caddis fly contains two allergens, while another contains only one.

Feinberg,[5] in testing a series of 225 persons sensitive to ragweed with pyrethrum extract, obtained positive reactions in 46.2 per cent. In testing a series of 113 patients not sensitive to ragweed, a positive reaction of mild grade was obtained in only 1 instance. Feinberg concludes that there is a group similarity between pyrethrum and ragweed pollen.

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2. Swineford, Oscar, and Weinberg, H.: Extreme Hypersensitiveness to Heat, J. Allergy **6**:530, 1933.

3. Duke, W. W.: Soy Bean as a Possible Important Source of Allergy, J. Allergy **5**:300, 1934.

4. Osgood, Howard: Comparison of Reagins to Separate Species of Caddis Fly, J. Allergy **5**:367, 1934.

5. Feinberg, S. M.: Pyrethrum Sensitization: Its Importance and Relation to Pollen Allergy, J. A. M. A. **102**:1557 (May 12) 1934.

Rowe [6] finds that camomile (Anthemis cotula) is a skin irritant to practically every one except during infancy. Certain persons, however, are more sensitive than others, and dermatitis venenata of varying degrees of severity will develop on contact with the weed. The substance responsible for the reaction is an oil or resin.

Tedstrom [7] reports a case of urticaria following the transfusion of blood from a donor who was suffering from hives and who gave a history of urticaria after the eating of strawberries. The transfusion was repeated later when the donor did not have urticaria. No such reaction followed the second transfusion. The author believes that the reaction was due to the transfer of histamine-like bodies from the donor to the recipient.

Greenhouse and Sulzberger [8] report a case of eczematous dermatitis caused by a perennial weed, tansy.

Harville [9] reports contact dermatitis due to the bleeding heart.

DIAGNOSTIC STUDIES

Rudolph and Cohen,[10] in an effort to discover the cause of perennial allergic rhinitis in patients who gave negative reactions to tests as ordinarily applied, placed dry pollen or epithelial materials on cotton pledgets and applied these directly to the turbinate membranes, leaving them in situ for about twenty minutes. They obtained positive reactions to one or several substances in a sufficient number of cases to improve the results of treatment 40 per cent in 19 persons who had failed to obtain relief by other methods. They suggest the use of this method in all cases in which cutaneous reactions are flatly negative.

Kern and Schenck,[11] studying the etiology of nasal polypi, state that they are extremely common in allergic disease of the respiratory tract. They state that in nonallergic disease of the respiratory tract mucous polypi are relatively rare, even in the presence of chronic infection of the sinuses. Polypi were found to be especially common in perennial cases; in fact, the authors conclude that the finding of nasal polypi warrants an assumption that the patient comes from an allergic

6. Rowe, A. H.: Camomile (Anthemis Cotula) as a Skin Irritant, J. Allergy **5**:383, 1934.

7. Tedstrom, M. K.: Active Transmission of Urticaria by Blood Transfusion, J. Allergy **5**:303, 1934.

8. Greenhouse, C. A., and Sulzberger, Marion B.: The Common Weed Tansy (Tenacetum Vulgare) as a Cause of Eczematous Dermatitis, J. Allergy **6**:523, 1933.

9. Harville, C. H.: Contact Dermatitis Due to a Common Plant, J. Allergy **6**:527, 1933.

10. Rudolph, J. A., and Cohen, M. B.: Vasomotor Rhinitis with Negative Skin Tests: A Local Nasal Allergy, J. Allergy **5**:476, 1934.

11. Kern, R. A., and Schenck, H. P.: Allergy a Constant Factor in the Etiology of So-Called Mucous Nasal Polyps, J. Allergy **5**:485, 1934.

strain. They state further that treatment for nasal polypi is likely to fail unless the element of allergy is taken into account.

Hansel [12] reports on a study of the cytology of nasal secretions which is in practical harmony with former observations which have been made along this line. He observed a large proportion of eosinophils in the secretions obtained from allergic noses and, in addition, the presence of neutrophils when there was evidence of superimposed infection. Hyperplasia and polyposis, with the persistent absence of eosinophils, prove, he states, that the condition is nonallergic.

Walzer and Grolnick [13] report on a study of papular urticaria and prurigo mitis in relationship to allergy. They found that 40 per cent of the patients with the former condition and all of those with the latter condition gave a family history of atopy. Positive cutaneous reactions were obtained in the cases of urticaria in which a family history of atopy was obtained. An increased eosinophil count is usually associated with positive cutaneous reactions. Neither specific nor nonspecific therapy was of value in the treatment of the conditions.

PATHOGENESIS OF ALLERGY

Simon and Rackemann [14] report on the development of hypersensitiveness in man following the intradermal injection of antigen and following the absorption of antigen through the nasal mucous membranes. They refer to the known fact that large quantities of serum injected into man may be followed not only by serum disease but by the development of cutaneous sensitiveness, which gives rise to positive reactions if the serum is suitably applied. They refer also to the fact that Hooker [15] and also Park [16] have shown that the small amount of horse serum in toxin-antitoxin treatment is sufficient to cause cutaneous sensitiveness in the person, so that thereafter appropriate application of horse serum will give positive cutaneous reactions. Tuft [17] finds that 27.9 per cent of children develop a cutaneous sensitiveness to horse

12. Hansel, F. K.: Observations on the Cytology of the Secretions in Allergy of the Nose and Paranasal Sinuses, J. Allergy **5**:357, 1934.

13. Walzer, A., and Grolnick, M.: The Relation of Papular Urticaria and Prurigo Mitis to Allergy, J. Allergy **5**:240, 1934.

14. Simon, F. A., and Rackemann, F. M.: The Development of Hypersensitiveness in Man: I. Following Intradermal Injection of the Antigen, J. Allergy **5**: 439, 1934.

15. Hooker, S. B.: Human Hypersensitiveness Induced by Very Small Amounts of Horse Serum, J. Immunol. **9**:7, 1924.

16. Park, W. H.: Is Serum Anaphylaxis a Danger of Sufficient Importance to Limit Our Use of Protective Sera in the Treatment or Prevention of Disease? Tr. A. Am. Physicians **28**:95, 1913.

17. Tuft, L.: Serum Sensitiveness After Toxin-Antitoxin, J. Allergy **3**:235, 1932.

serum after treatment with the Schick diphtheria toxin-antitoxin mixture. Jones and Mote [18] found that patients given repeated injections of rabbit serum began giving positive cutaneous reactions of the inflammatory type, resembling the tuberculin reaction, and later, after repeated doses, reactions typical of hives. Simon and Rackemann, in their work, compared the effect of repeated small doses of guinea-pig serum on the sensitivity of the skin in two sets of persons, the atopic class and the nonatopic class. In each set they regularly, and with about the same degree of intensity, produced cutaneous sensitiveness by one or several doses of serum of dilutions between 1 : 10 and 1 : 1,000. Cutaneous sensitiveness so induced gave rise, after a small number of doses, to the tuberculin type of reaction, delayed in its appearance, and after a greater number of doses, to the immediate production of a wheal. Even with high dilutions of the serum (between 1 : 100 and 1 : 100,000) patients of both classes had reactions which varied in the degree of sensitiveness but did not seem to be definitely different one from the other. In other words, the atopic person did not react more intensely than the normal person. Sensitiveness so induced lasted for months, but the intensity of the cutaneous reaction was inclined to lessen gradually. No constitutional reactions were observed, nor were the reactions at any time so intensely positive as is observed in patients who are naturally hypersensitive to pollen and to other substances.

In a second paper [19] the same writers describe the induction of sensitiveness both in the nasal membranes and in the skin brought about by the application of guinea-pig serum to the nasal membranes one or several times. They found that nasal reactions resembling hay fever followed the reapplication of guinea-pig serum to the nasal membranes. Also, that after several applications cutaneous sensitiveness developed. The symptoms produced consisted of nasal obstruction with serious discharge and sneezing, which began from six to twenty-four hours after the application and lasted several days.

The foregoing experimental results harmonize closely with facts as observed clinically, such as the development of sensitiveness to such agents as orris, grain dust, castor bean dust, soy bean dust and other substances among persons who are thrown by habit or occupation into natural contact with them. These results are at variance with earlier observations of other authors, which lead to a general conclusion that it was difficult or impossible to sensitize persons artificially.

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18. Jones, T. D., and Mote, J. R.: The Phases of Foreign Protein Sensitization in Human Beings, New England J. Med. **210**:120, 1934.

19. Simon, F. A., and Rackemann, F. M.: The Development of Hypersensitiveness in Man: Absorption of the Antigen Through the Nasal Mucous Membrane, J. Allergy **5**:451, 1934.

It can be assumed now, without question, that persons of both the atopic and the nonatopic family can be sensitized either artificially or by natural contact with alien proteins.

Fisher [20] reports a case of sensitiveness to bees with tolerance obtained through treatment with bee extract, which is interesting from several points of view. The patient lived in the neighborhood of bee hives after 1927. During the first year she was stung frequently with no ill effect beyond that experienced by a normal person. However, during the following year she recalled attacks of asthma and urticaria. Later, sensitiveness developed to such a degree that the proximity of clothes worn by men handling bees caused asthma, and contact with the hands of persons handling bees caused local urticaria. Also, bee sting would precipitate dangerous reactions. The patient was treated with infinitesimal gradually ascending doses of bee extract, with the result that bee stings produced only a trivial illness.

EFFECTS OF ALLERGY

Shookhoff and Lieberman [21] report the finding of symptoms of angina pectoris in 3 patients caused by the ingestion of acetylsalicylic acid. In 2 cases the attacks were accompanied by urticaria.

Black [22] reports reduced blood sugar and an increase in sugar tolerance in more than half of the patients with hay fever and asthma tested during a period when they were having allergic symptoms.

Rinkel [23] reports on a study of headache due to allergy and mentions three separate types: the migraine type, a type associated with allergy of the nose and sinuses and a type occurring as part of a general allergic reaction. He states that the migraine type constitutes less than 2 per cent of the entire group. He found that the reactions to cutaneous tests were positive in about 30 per cent of cases, and that clinical tests were more valuable in diagnosis. He treats his patients with elimination diets and symptomatic therapy.

STUDIES OF TREATMENT

Bayer [24] reports an interesting example of rapid desensitization in a case of allergy to insulin. The patient, who had been on insulin ther-

20. Fisher, D. C.: Hypersensitivity to Bees Successfully Treated with Whole Bee Extract, J. Allergy **5**:519, 1934.

21. Shookhoff, C., and Lieberman, D. L.: Angina Pectoris Syndrome Activated by Ragweed Sensitivity in a Patient With Coronary Vessel Sclerosis: Case Report, J. Allergy **6**:513, 1933.

22. Black, J. H.: Blood Sugar in Allergic Persons, Texas State J. Med. **29**: 257, 1933.

23. Rinkel, H. J.: Headaches Due to Allergy, J. Allergy **5**:318, 1934.

24. Bayer, L. M.: Desensitization to Insulin Allergy, J. A. M. A. **102**:23 (June 9) 1934.

apy, became so sensitive that a 5 unit dose was followed within twenty minutes by violent abdominal cramps, macular itching rash and a sense of choking. He found 1/1,000 unit could be tolerated at this point. He gave injections in ascending dosage at intervals of one-half hour without ill effect until tolerance was pushed up to a point where 5 units could be given. The dose was eventually increased to 10 or 15 units daily and could be given without ill effect except for occasional slight attacks of hives.

Colmes and Rackemann [25] report on changes in cutaneous reactions following specific pollen therapy. They state that good results in treatment are accompanied by a reduction in the positiveness of cutaneous reactions in the majority of patients. However, good clinical results in the absence of any change in cutaneous reactions were observed in 18 per cent of cases, and poor results were observed in the presence of reduced cutaneous reactions in 11 per cent of cases. They conclude that relief from symptoms and reduction in cutaneous reactions did not occur on a common basis, and that cutaneous tests as now performed are only a crude index of the degree of clinical sensitiveness.

Fineman [26] reports on a study of four cases of asthma in which suprarenal cortex extract was given subcutaneously from three to four times a week for one month, and thereafter once or twice weekly. One of the patients showed marked improvement in the asthma, 1 moderate improvement, 1 slight improvement and 1 no improvement. In 3 of the patients there was a decided increase in strength and gain in weight, averaging 4½ pounds (2 Kg.). The general condition of these patients seemed definitely better. No appreciable change in blood pressure or pulse rate was observed.

Lichtenstein [27] reports on intravenous pollen therapy which he carried out without mortality in 6 patients. He states that this method of therapy must be considered an experiment to be used only by those who have considerable experience in allergy.

He advances a number of reasons why this should be theoretically the method of choice. For example, he states that the dose can be delivered in a more scientific way than by subcutaneous injection because the allergic reaction itself at the site of inoculation delays the rate of absorption and delivery of the extract to the blood stream. Because of the gross variance in different persons in the degree to which they react locally, the rate of absorption varies. The effect produced by a given dose of extract, therefore, is not the same in all cases. All the

25. Colmes, A., and Rackemann, F. M.: Further Observations on the Changes in Skin Tests Following Specific Pollen Therapy, J. Allergy **6**:473, 1933.

26. Fineman, A. H.: The Use of Suprarenal Cortex Extract in the Treatment of Bronchial Asthma, J. Allergy **4**:182, 1933.

27. Lichtenstein, M. R.: Intravenous Pollen Therapy, J. Allergy **5**:230, 1934.

reasons mentioned by Lichtenstein in defense of this method of treatment, however, do not justify its use at the present time, since the reactions produced were frequently severe though controllable in the six patients whom he treated. Unfortunately, cerebral tissues and cardiac tissues may take part in the allergic reaction with alarming results. Even though these can be controlled, as a rule, with epinephrine, their frequent occurrence with intravenous therapy would soon discredit the use of the method. Lichtenstein errs in assuming that reactions from subcutaneous injections are unavoidable. While this may be true in the hands of inexperienced physicians, it is not so in the hands of those who take all the precautions possible to make their method of administration fool-proof. Lichtenstein made an interesting observation of theoretical importance in finding that when too concentrated material was injected closure of the vein resulted owing to allergic swelling of the wall of the vessel.

Phillips [28] reports on a six year study of his intradermal and coseasonal method of treatment which was referred to in an earlier issue of the ARCHIVES. The point of interest is that Phillips begins treatment with weak dilutions of pollen extract given intradermally with increase in dosage and time interval gaged according to the patient's reaction. He states that with this method of treatment he usually gains practical relief in the gross majority of cases within a period of days. Satisfactory relief was obtained in 96.6 per cent of 322 patients, all of whom could be observed by him personally.

The method as described by Phillips is well worthy of reading and has been the most effective method I have ever used both in percentage of favorable results and in the quickness in which favorable results can be obtained. At present, however, I use a shock proof method, which is less rapidly effective but safe from a chance of reaction.

Warner and McGregor [29] report that following 31 operations on the antrums of patients who had asthma, relief was obtained for periods varying from two weeks to a little over two years. The greatest benefit was derived by the patients who had had asthma only a short time and who presented a very much thickened membrane.

IONIZATION

The term ionization designates a method of treatment which consists essentially of the passing of a direct electrical current through an electrolite into the nasal structures. Although this idea was introduced a

28. Phillips, E. W.: Intradermal Pollen Therapy During Attack, J. Allergy **5**:29, 1933.

29. Warner, W. P., and McGregor, B.: Effect of Surgery on Bronchitic Asthma, J. Laryng. & Otol. **48**:585, 1933.

number of years ago, the method of treatment is at best in an experimental stage and will remain so for a number of years if actual merit justifies its scientific study.

The present-day literature which I have read on ionization is characterized by rather bold statements backed up by little published clinical, experimental, pathologic or immunologic data which can stand up under constructive review. In spite of this, however, the medical profession has been fairly well supplied with machinery for ionization treatment.

It seems to me that premature commercial exploitation of a remedy based on apparent results in a few cases always has been and always will be a menace to the normal, sane practice of medicine. More remedies have been heralded as cures for allergy than for almost any other disease known to medical science. Also, more old remedies have been taken up and rediscarded. There is actually a sound reason for this circumstance. Allergy is caused as a rule by agents which neither physician nor patient can see. Both are inclined to blame the source of the trouble on conspicuous agents which they can see. They also falsely acclaim remedies which are used when patients unwittingly escape unseen noxious contacts and then falsely condemn remedies used when they again meet with the agents responsible for their illness.

It is almost impossible for a doctor to evaluate the therapeutic value of any remedy he uses unless he actually knows the one or several agents which make his patient ill, and whether or not the patient is actually in contact or out of contact with such agents. This sounds easy, but it is actually extraordinarily difficult for more reasons than one.

Since pollen is the most common single cause of nasal allergy, it is necessary for a doctor to know daily the pollen content of the air before he can evaluate a given method of treatment for hay fever and asthma caused by pollen. During years of extreme drouth, such as occurred in the Middle West in 1930 and 1934, the pollen content of the air was reduced to a small fraction of what is found in years when rainfall is favorable to vegetation. Furthermore, dryness of the air, which has a potent effect on evaporation and the cooling of nasal membranes, is increased in years of drouth, so that this, too, has a favorable influence on patients with wet noses. This makes any method of treatment work better.

Kansas City and similar botanical areas have not had what can be called a normal pollen year, or rather a full pollen year, since 1927. The blooming of the spring trees in this and similar botanical districts, has, with certain exceptions, been stopped by frosts. The grasses have been very irregular in their pollination, as a result of unfavorable weather conditions, and the fall weeds have never reached the heights of pollination observed in 1926 and 1927. The fact is that in 1926 and 1927 pollen counts running as high as 2,000 and 4,000 (per square half inch surface)

were found frequently in the spring months, and almost daily or several times weekly for a number of weeks after August 25. Since 1927 pollen counts have rarely exceeded 1,000 and, as a general rule, have been far below 500, even at times of maximum pollination. During the drouth years 1930 and 1934, the pollen counts have actually ranged on a majority of days below 100 to 300. During the summer of 1934 there was rarely enough pollen on the slides to count. Facts such as these have to be known to physicians in a given district before they can evaluate their therapeutic methods, for, regardless of methods, during favorable years favorable results will be obtained by almost any common sense treatment or by no treatment.

In my own work in 1934, more than 150 patients sensitive to ragweed were treated by education concerning avoidance of specific allergens, by specific treatment with allergens which could not be avoided and by common sense medical measures, and with the aid of otolaryngologists. I do not know of one of these patients who suffered materially this year from pollen disease. The fact is, only 7 patients appeared in my office this year with symptoms of hay fever of even moderate grade, and these were relieved almost completely and promptly by mild methods of treatment. I give credit for these unusual results this year to the drying up of vegetation and to dry air rather than to unusual effectiveness of the treatment used.

The majority of cases of both seasonal and perennial allergy not caused by pollen are traceable to inhalant allergens, foods, heat, effort, cold, light or infection, and usually to more than one agent.

It is not my object in this review to praise, to condemn or to be indifferent to the treatment called ionization. It will find its place in the treatment of allergy if it has one. However, the premature commercial exploitation of any method in an experimental stage is bad for the method if the method is good, and bad for the medical profession if the method is bad. Intimations of permanent cure of true hereditary allergy by one or several treatments of any sort is out of harmony with the present-day knowledge of the disease.

Abstracts from Current Literature

Ear

LABYRINTHINE TESTS AND THEIR AID TO DIAGNOSIS. A. R. TWEEDIE, J. Laryng. & Otol. **49**:160 (March) 1934.

Tweedie believes that the cold caloric and galvanic tests are superior to rotation in the clinical investigation of labyrinthine function. From the complicated observations of nystagmus three fundamental factors emerge: (1) the initial deviation, (2) the secondary involuntary reflex and (3) the true volitional effort. The author suggests that in nystagmus a simple primitive reflex which may become an extremely complex proposition is concerned. More is known of the physiologic results of artificial stimuli than of the effects of pathologic lesions. Since the physiologic reactions to the caloric and galvanic tests are well known and the tests can be graduated fairly accurately and can be restricted to one side, they are the most useful. These tests are particularly applicable in testing vestibular function in noninflammatory conditions, but they are also of use in determining the degree and extent of remaining function in postinflammatory conditions. Attempts should still be made to assess the influence of spontaneous nystagmus in cases of acute otitis media. The author feels that the "blinding" method of de Kleyn may increase the knowledge of nystagmus in this condition.

EDITOR'S ABSTRACT.

SECONDARY THIERSCH GRAFTING THROUGH THE MEATUS OF THE MASTOID CAVITY AFTER RADICAL OPERATION. W. I. DAGGETT and G. H. BATEMAN, J. Laryng. & Otol. **49**:169 (March) 1934.

Daggett and Bateman believe that the logical time for inserting a graft in the mastoid cavity after radical operation is at the end of two weeks. Two objects are essential: rapid and complete epithelialization and the avoidance of a large number of or painful dressings. After trying several modifications the authors devised a technic which they consider satisfactory. This is summarized in seven steps: (1) operation, with special attention to fashioning a wide meatus; (2) on the seventh day changing of the dressing, under nitrogen monoxide anesthesia; (3) from the seventh to the fourteenth day, changing of the dressing and syringing of the cavity when necessary; (4) on the fourteenth day, under anesthesia with a barbituric acid derivative, insertion of the graft in the cavity and pouring in of wax; (5) on the fifteenth or sixteenth day discharge of the patient from the hospital, the top dressing being changed when necessary until the twenty-eighth day; (6) on the twenty-eighth day, removal of the wax and insufflation of powder, and (7) periodic inspection of the cavity as circumstances dictate.

EDITOR'S ABSTRACT.

STATISTICAL COMPARISON OF THE RESULTS OF PRIMARY CLOSURE OF A RETRO-AURICULAR WOUND AND OPEN HANDLING OF MASTOIDITIS. H. WRANGEL, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:351 (Dec. 20) 1933.

Since 1930 the mastoid operation of Bárány has been used in Lund. The closure has been accomplished by primary suture of the periosteum and skin, a cigaret drain being left at the lower end of the incision. The drain is removed after two or three days. Wrangel has compared the results of this method with those secured by open drainage. From 1925 to 1932, 688 patients with mastoiditis were treated at the clinic, 295 by open drainage without complication, 82 by open drainage with resulting complications, 100 by primary closure with healing

by primary union and 11 by primary closure with healing by second intention. The remaining patients were treated by radical operations for acute mastoiditis, were transferred to other divisions after the operation, were not operated on, were subjected to exploratory examination, had recurrence after open treatment, had mastoiditis as a complication of scarlet fever or tuberculous mastoiditis, had postoperative complications after open treatment or had intercurrent infections. On analyzing the results the author found that the entire period of handling lasted on the average five weeks less when primary closure was used. Certain disadvantages of primary closure were evident, however. In 10 per cent of the cases a purulent infection occurred in the edge of the wound or in the soft tissues, so that the treatment thereafter had to be open. In these cases the duration of treatment was longer than in those in which the original treatment was open. There seemed also to be a higher percentage of intracranial complications in the cases of treatment by primary closure, though the author does not feel that he has observed enough cases to warrant final conclusions in this comparative study.

EDITOR'S ABSTRACT.

REPORT ON THE FIRST TWENTY CASES IN WHICH OPERATION WAS PERFORMED FOR OTOSCLEROSIS. H. ROLLIN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **138**:6 (March) 1934.

The operative treatment for otosclerosis described by Rollin is based on the etiologic stasis theory of Wittmaack. The incision begins about 2 cm. above the upper insertion of the pinna and extends horizontally from the hair line. It passes through the skin and muscle to the bone for about 6 cm. so that the upper pinnal insertion lies exactly in the middle. After detaching the periosteum and stretching the incision (the wound speculum of Wittmaack is used for this), a small trephine opening is made in the bone with a hammer and chisel. The opening is enlarged semicircularly with a bone forceps to about the size of a mandarin slice so that the base of the incision runs parallel to and near the base of the skull. The dura is separated from the anterior surface of the temporal bone by means of an elevator, and the dura-covered cerebrum lifted a little from the bone with a thin, flexible brain spatula. In this small space blunt dissection is carried along the upper pyramidal surface over the eminentia arcuata until the exit of the nervus petrosus superficialis major from the skull is reached. This nerve with its accompanying vein is bluntly dissected. The separation of the dura from the bone is performed with the small elevator usually used in operations on the septum. The severed temporal muscle is united to its fascia with catgut and the skin closed with silk sutures. If there was a difference in hearing between the ears the operation was performed on the side on which hearing was better. If the hearing was about the same it was performed on the ear which presented the greatest subjective symptoms. In only one instance was there a serious complication. This was facial paresis. In two instances subcutaneous hematomas formed, which, however, did not prevent primary wound closure. The cases of eighteen patients and the operations are described in some detail. In one of these an adhesive process rather than otosclerosis was probably the cause of the symptoms. Of the others, objective evidence of postoperative decrease in hearing in the ear operated on occurred in only one, and progressive deafness in the ear which had not been operated on, in five. An improvement in hearing occurred in two ears which were operated on and in none of those not operated on. Subjectively all of the patients noticed increasing hearing defects in the period immediately preceding operation. After the operation, seven patients thought that there was a pause in the progress of the hearing defect, and five thought that there was a slight improvement. Three patients claimed that there was beginning improvement, and one beginning cessation of progressive deafness. One patient claimed that her hearing had become worse in spite of the operation. Five patients spontaneously requested an operation on the other ear. Twelve patients complained of aural noises before the operation. Ten of these stated that there was remission following the operation. The author feels

that the first principle of not injuring the patient by attempted treatment is complied with, and he recommends this procedure for further trial.

EDITOR'S ABSTRACT.

BEHAVIOR OF THE TEMPORAL BONE IN CYSTICERCOSIS OF THE BRAIN AND IN CARCINOMATOUS MENINGITIS. H. BRUNNER, Monatschr. f. Ohrenh. **68**:257 (March) 1934.

In a case of secondary meningeal carcinomatosis Brunner found bilateral infiltration of the arachnoid sheath of the eighth and facial nerves with seal ring cells and lymphocytes, and splintering of both branches of the eighth nerve by infiltrating cells. Atrophy of the spinal ganglion and nerve of the vestibule and basal convolutions, with infiltration of this ganglion with seal ring cells, and an otosclerotic focus in the foramen rotundum were also found bilaterally. On the right side atypical arteries in the posterior sheath of the pyramid and a small amount of serous fluid in the cochlear canal of the lower convolution were found. On the left side Brunner found an otosclerotic area on the floor of the fundus of the inner ear passage, atypical connective tissue extending from the horizontal crista to the tympanic tegmen, dilatation and engorgement of the blood vessels of the inner ear, fresh hemorrhages in the nerve canals, in the scalae, spiral ligament and macula and a large amount of serous fluid in the cochlea, vestibule and labyrinth. This large number of changes established the fact that the temporal bone had an abnormal development. The author also reports a case of cysticercosis of the brain.

In concluding this comparative study of the behavior of the inner ear in chronic increased cerebral pressure, definite differentiation between local and general pressure must be drawn. General pressure causes no microscopically observable changes in the inner ear, while acute fluctuations of the general intracerebral pressure may produce changes in the inner ear comparable to commotio cerebri. On the contrary, chronic local cerebral pressure as a rule produces changes in the inner ear which can be summed up as congestive hydrops. This congestive hydrops manifests itself frequently in hemorrhages and transudates but as a rule does not develop to the production of secondary changes (connective tissue formation and atrophy of the sensory epithelium). When, therefore, definite symptoms of peripheral changes in the ear occur in diseases of the cerebellopontine angle, only in rare instances are they due to congestive hydrops. If in such cases one thinks of a blastomatose condition of the eighth nerve in the cerebellopontine angle or of some other disease of the ear (e. g., otosclerosis) the diagnosis will more often be correct.

EDITOR'S ABSTRACT.

INTERFERENCE PHENOMENON IN POSTROTATORY NYSTAGMUS. K. ARSLAN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:109 (Feb. 10) 1934.

Arslan carried out a series of observations on nystagmus after horizontal rotation. In the first group two patients were rotated in a Tönnies chair at such a rate that in about three minutes an angular speed of 180 degrees per second was reached. The chair was stopped mechanically, and the duration and number of postnystagmoid motions were observed. Six minutes after the beginning of the first rotation it was repeated. The procedure was carried out ten or twelve times on each patient. Twelve similar series of rotations were performed so that for each patient more than one hundred and twenty individual observations were made. In the course of the series decided deviations from the normal value were found following both the first and the second rotation. In general, a tendency to diminution of nystagmus after the later rotations was observed, though this was not constant. In a second group of observations normal persons were subjected to numerous repeated rotations. After a fixed interval of five seconds they were rotated in the opposite direction and after variable intervals again retarded. The single physical stimulus was always constant. Variable and interesting changes in the direction, duration and number of jerks were observed following these

rotations. Abnormally long latent periods were also seen. Further rotation was employed in which the initial velocity as well as the final retardation were in excess of the threshold value with which the interval between the stimuli was varied. Here the postrotatory nystagmus was determined by the interval. The author believes that all of the observed changes of postrotatory nystagmus can be attributed to interference phenomena in the central nervous system (vestibular nucleus and so on).

EDITOR'S ABSTRACT.

HEALING PROCESS IN OLD LABYRINTHINE FRACTURES: ROENTGENOGRAPHIC DEMONSTRATION. K. ULRICH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:263 (March 12) 1934.

In August 1932 Ulrich attempted to reach all of the patients of the Zurich Ear Clinic who had shown definite clinical and roentgenologic evidence of transverse fractures of the labyrinth. Of the eight patients obtained for roentgenographic examination three had pure transverse fractures, three isolated cochlear fractures and two combined transverse and longitudinal fractures. The small number of patients does not allow any conclusion to be drawn as to the relative frequency of the different types of fractures. The author found, however, that in all cases, in spite of a lapse of from two to twelve years since the accident, the labyrinthine fracture was still recognizable in the roentgenogram. Three differences in the appearance of the old fracture from that seen in the early roentgenogram were observed: First, the fracture line often no longer extended through the upper pyramidal edge but first became evident in the inner part of the bone. Second, the broken ends were not so sharp but seemed somewhat rounded. Third, the fracture line was better seen in the inner portion of the petrosa than at the periphery. He concludes that new bone production begins in the periosteal region and eventually extends to the endosteum but that only connective tissue healing occurs in the endochondral labyrinth, thereby allowing the fracture line to remain visible in the roentgenogram.

EDITOR'S ABSTRACT.

CLINICAL OBSERVATION ON HEARING AND EQUILIBRIUM APPARATUS IN METAPOIETIC BONE DISEASES. (OSTEODYSTROPHY OF PAGET AND OSTEODYSTROPHY OF RECKLINGHAUSEN). H. BARTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:305 (March 12) 1934.

Barth describes four patients with osteodystrophy of either the Paget or the Recklinghausen type who had functional disturbances of hearing and equilibration. He also summarized twenty-eight cases from the literature in which known disturbances of hearing and equilibrium were associated with metapoietic diseases of the bone. Up to the present time, it does not appear that functional disturbances of the ear are especially frequent. It is necessary, however, that in these disease pictures more attention should be paid to the otologic side. It is accepted that the hearing and equilibrium apparatus may be injured in the osteodystrophy of Recklinghausen as well as in that of Paget. The observations of hearing tests showed pure inner ear deafness in six cases, combined inner and middle ear deafness in eight and simple middle ear deafness in one case. These statistics are subject to the criticism that the cases of inner ear deafness may be due to old age and entirely independent of the bone condition. Attacks of dizziness play a greater rôle in the history of these patients than difficulty of hearing.

EDITOR'S ABSTRACT.

A CASE OF ENTOTIC NOISES. D. C. EBSKOV, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folio oto-laryng.) **25**:45, 1934.

Ebskov reports an unusual case of a woman, aged 37, who for eight years heard noise in the right ear. The noise became constant and was sometimes rhythmic and sometimes entirely irregular. It was uninfluenced by external factors, such

as sleepiness, the weather and the menses. It was so loud that it disturbed sleep and could be heard by others when listening close to her ear. Light pressure in and around the right ear was noticeable but produced no real pain. In the left ear, hearing was good, and there were no symptoms. Examination revealed a slightly retracted tympanic membrane. Examination with a convex lens showed a pulsating light reflex. In listening to the right ear, fine strokes, sometimes regular, sometimes irregular, could be heard. Their frequency varied from 0 to 100 a minute. The remaining local and general observations were essentially normal. Entotic noises may be due to blood vessels, mucous membrane or muscles. The author feels that the first two factors were eliminated in this case because of the normal general condition. Contraction of the tensor tympani, the stapedius or the tensor veli palatini muscle may produce such sounds. Which muscle was involved in the present case was hard to determine, though the choice probably lay between the first two. The patient was treated with diathermy, Finsen's baths, a galvanic current, digital massage of the tubal orifice and insertion of a tubal catheter and of a tubal catheter with intratympanic injection of 4 per cent procaine hydrochloride. The patient was finally somewhat improved.

EDITOR'S ABSTRACT.

EPIDERMAL TRANSPLANTATION (PONTOPPIDAN METHOD) IN RADICAL OPERATIONS ON THE MIDDLE EAR. R. VON MAGNUS, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folio oto-laryng.) **25**:48, 1934.

Von Magnus states that skin grafting by the Pontoppidan method has been used by him as a routine procedure for two years in cases of radical operations on the middle ear. The radical operation is done by the usual method under ether narcosis. Since it is important to have a bloodless field, 1 cc. of procaine hydrochloride is injected into the aural appendages and canal after induction of the narcosis. This tends to give a bloodless field. After the operation is finished the cavity is cleaned with oxydol (hydrogen dioxide) and irrigated with water and a 2 per cent solution of phenol and physiologic solution of sodium chloride. To prevent later hemorrhage, an epinephrine tampon is placed in the cavity. The skin of the thigh is prepared by washing with a 2 per cent solution of phenol and physiologic solution of sodium chloride. One or two large, thin strips are cut and placed against a gauze strip previously impregnated with hydrous wool fat with the epidermis against the hydrous wool fat and the corium outward. After the epinephrine tampon is removed the skin and gauze are introduced into the cavity of the operation and pressed against the wall with little pieces of gauze. A sterile dressing is then applied. From five to eight days thereafter, the dressing is changed for the first time, and all of the gauze is removed. The author has performed this procedure on thirteen patients. Excluding the two cases in which operation was performed most recently, in which the wounds have not yet entirely healed, the average duration for complete epidermization was forty-seven days, with a variation of from twenty-two to seventy-eight days. In eleven other cases of radical operation, chosen at random, without transplantation, the average duration of after-care was eighty-four days, with variations of from fifty-eight to one hundred and eighty days. The author believes that this method allows a much shorter postoperative observation period, an almost completely painless changing of dressings, the possibility of better results in hearing, and finally a cavity which is always more spacious than that obtained when transplantation is not employed.

EDITOR'S ABSTRACT.

Pharynx

PROBLEM OF TONSILS. S. PELLER, Monatschr. f. Ohrenh. **68**:417 (April) 1934.

Peller reached certain conclusions as to the function and significance of the tonsils by making statistical comparisons on the constitution and state of the tonsils of a large number of persons. The tonsils act as an inhibitory factor in growth.

They inhibit height, weight and thoracic expansion. The degree of inhibition is great enough to determine statistically but not enough for casuistry. The tonsils lengthen the period and decrease the rate of growth. Inhibitory impulses are increased by hypertrophied tonsils. Chronic tonsillitis apparently has no effect on the growth-inhibitory factor. Tonsillectomy affects growth in a manner independent of the factors of time, social position and age. This is a lasting effect which is still shown years after the tonsillectomy. The tonsils are an endocrine organ whose function is not exhausted by or identical with that of the lymphatic system. Otherwise the surprising, even if only statistical, established and demonstrable deficiency phenomena would not be clear. Tonsillectomized girls menstruate about a year earlier than those with hyperplastic tonsils and mature earlier than the average girl. There is a frequency relation between the color of the skin and hair and tonsillar hypertrophy. Blondes under similar circumstances present hypertrophy more frequently and are tonsillectomized more frequently than brunettes and darkly pigmented persons. The tonsils are a useful factor in the photochemical defense reaction of the skin. The author concludes that future research must bring out further relationships and explain those which are described here.

EDITOR'S ABSTRACT.

SUBACUTE AND CHRONIC CERVICAL LYMPHADENITIS AS A DIAGNOSTIC SIGN OF PRIMARY TONSILLAR TUBERCULOSIS. E. SCHLITTLER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:400 (April 6) 1934.

A review of the cases of primary tonsillar tuberculosis confirmed by necropsy shows that a swelling of the lymph nodes draining the tonsils occurs in the neighborhood of the angle of the jaw. Schlittler reports that in ninety-eight cases in which the patients had chronic or subacute lymphadenitis at the angle of the jaw definite microscopic tuberculosis was demonstrated in the removed tonsils forty-eight times. Primary tonsillar tuberculosis is therefore the diagnosis of choice in the presence of this type of adenopathy. In these forty-eight cases of histologically proved tonsillar tuberculosis, a pharyngeal tonsil was the seat of the lesion seven times, a pharyngeal and a palatine tonsil six times, both palatine tonsils seven times, and one palatine tonsil twenty-eight times. Several features of the condition in these forty-eight cases speak for the primary involvement of the tonsil: the absence of open pulmonary tuberculosis or tuberculosis of other organs; the further behavior of the patients after removal of the tonsils; the strict localization of enlarged lymph nodes to the region draining the tonsils, and finally the relative frequency of involvement of only one tonsil (73 per cent). The author believes that in the rest of the ninety-eight cases, in which no tuberculous changes were found in the tonsils, the primary factor might still be tuberculous, but the condition had healed to such an extent that the original lesion was not found. He finds evidence for this view in the decreasing number of cases of this type in which he and his associates are unable to find tuberculosis of the tonsil after removal.

EDITOR'S ABSTRACT.

THE ROENTGEN PICTURE OF THE EPIPHARYNX OF CHILDREN UNDER NORMAL CONDITIONS AND WHEN ADENOID VEGETATIONS EXIST. KARL ERICH GROTH, Acta radiol. **14**:463 (Oct.) 1933.

In certain cases, diagnosis by palpation and rhinoscopy is difficult or unjustifiable. Roentgen examination skilfully done gives a clear picture of the size of the adenoid and of the space in the epipharynx and can be carried out, if necessary, during sleep. Lately Groth has examined patients with adenoids with the roentgen rays to discover to what extent the passage through the epipharynx is affected by different positions of the head and by sleep. The profile picture is best suited for the study of these relations. In a normal child in the upright position the passage is seen to be broad and spacious and affected only inconsiderably by different positions of the head. With adenoid vegetations in the epipharynx the

roentgen picture presents another appearance. A considerable swelling on the posterior pharyngeal wall represents the pharyngeal tonsils, and the soft palate appears as a shadowy band narrowing backward below the adenoid, with a thin lumen of air between, which indicates the narrowed epipharyngeal passage. The appearance is then practically the same whether the patient is in the recumbent or in the sitting position when the picture is taken.

VINTON, Brooklyn. [AM. J. DIS. CHILD.]

Larynx

PATHOGENESIS AND THERAPY OF VOCAL CORD NODULES. T. VON BAJKAY, Monatschr. f. Ohrenh. **68**:325 (March) 1934.

Nodules of the vocal cords apparently arise from chronic laryngeal catarrh, from overuse of the voice (e. g., singers' nodes) or from a poor technic of speech as a result of which the vocal cords are pressed together in an abnormal way. In Bajkay's series, "chorditis nodosa" occurred in 20 men and 70 women among the 28,000 patients seen in the clinic in the past years. There were 7 boys and 2 girls aged between 6 and 15 years, 2 men and 34 women between 16 and 25, 7 men and 32 women between 26 and 45, and 4 men and 2 women between 46 and 58. Seventeen male patients and 65 female patients had nodules of both vocal cords; 2 male patients and 2 female patients had nodules of the left cord, and 1 male patient and 3 female patients had nodules of the right cord. In many cases there were no symptoms, but in others there were severe disturbances, which sometimes resulted in loss of speech. The prognosis is good in general. The therapy is conservative or operative. The most important aspect of treatment is rest of the larynx. Local application of silver nitrate or strong silver protein sometimes gives good results. Iodides have been reported on favorably. When conservative treatment fails, intralaryngeal operative removal may be attempted with frequent satisfactory results.

EDITOR'S ABSTRACT.

OPERATIVE TREATMENT OF SO-CALLED DIAPHRAGM OF THE LARYNX. K. BECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:303 (March 12) 1934.

The case of a girl, aged 16, with shortness of breath on exertion and hoarseness due to a membranous formation in the larynx is described by Beck. After two unsuccessful attempts to relieve the condition, an external operation was performed. Under local anesthesia a collar incision was made (for cosmetic reasons) and the larynx opened. The membrane was pushed aside with the knife, and a rubber tube was placed in the trachea, extending to the lips of the larynx. This tube was allowed to remain three weeks, after which it was removed and the incision closed. Eleven months later the glottis remained free, and there was good scar formation on the anterior commissure. The patient was much improved, though the voice retained a hoarse note. The author feels that this method can be recommended.

EDITOR'S ABSTRACT.

Nose

ATROPHIC RHINITIS. J. ADAM, J. Laryng. & Otol. **49**:375 (June) 1934.

In analyzing 141 cases of atrophic rhinitis, Adam found that in at least 78 per cent the disorder began before puberty and in at least 42 per cent during the first seven years of life. These are the years during which the face and the nose and its accessory cavities are normally developing most quickly. This development is consequently retarded. When the disease begins in adult life, as it rarely does, the typical facies of atrophic rhinitis does not occur. The disease begins as inflammation of the nasal mucosa, and in at least half the cases, probably more than 66 per cent, there is sinusitis. This tends to keep the inflammation

alive and is often missed. The ethmoid is more often affected than is realized. This also applies to adenoids. The present custom of dealing with adenoids early has reduced the incidence of atrophic rhinitis. Another factor is deficiency in vitamins, especially vitamin A. This deficiency impairs defense against infection and lowers endocrine function. It may also impair nervous function. The mucosa reacts first by hyperplasia; later, owing to fibrosis, there are glandular atrophy and change from columnar epithelium to stratified epithelium; but if the sinusitis is conquered early enough by proper surgical and other measures there may be considerable recovery with disappearance of crusts and fetor. The bone of the thin lamellae of the turbinates and the ethmoid cells reacts by atrophy; that of the walls of the accessory cavities, by sclerotic thickening. Similar sclerosis is found in the mastoid processes of children with chronic suppuration of the middle ear. The paranasal sinuses may fail to reach their full development, with consequent facial modification.

EDITOR'S ABSTRACT.

NEURITIS DUE TO ALCOHOL AND TOBACCO AND MORPHOLOGIC CHANGES IN THE NOSE AND SINUSES. E. MORELLI, Ann. d'ocul. **170**:697 (Aug.) 1933.

Morelli observed morphologic changes in the nose and in the sinuses in cases of neuritis due to alcohol and tobacco.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

HEADACHE OF NASAL ORIGIN. SARGNON, Ann. d'ocul. **170**:699 (Aug.) 1933.

Sargnon devoted careful and critical study to headaches of nasal origin. He states that they are due to acquired lesions, particularly to anterior ones, such as traumatic, inflammatory, nervous or neoplastic lesions. He discusses the etiology and symptomatology of these lesions in relation to medical and to surgical treatment.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

MODIFICATIONS OF THE NASAL MUCOUS MEMBRANE FOLLOWING CERVICAL SYMPATHECTOMY: HISTOLOGIC AND EXPERIMENTAL STUDY. R. MOTTA, Ann. d'ocul. **170**:701 (Aug.) 1933.

Motta observed the nasal cavities after removal of the cervical sympathetic nerve in young rabbits (about 24 days old). He concludes that when the superior ganglion has been removed, unilateral sympathectomy provokes primarily (after eighteen hours) vasodilatation in the nasal mucous membrane of the opposite side and later a mild homolateral syndrome. Twenty-eight days after the simultaneous removal of the cervical sympathetic nerves on both sides, the mucous membrane on one side is velvety and the vessels are dilated, and the mucous membrane on the other side is ischemic and shrunken.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

MUCOCELE OF THE MAXILLARY SINUS. M. TAKANO, Acta soc. ophth. jap. **37**:15 (Feb.) 1933.

The usual mucoceles of the nasal sinuses generally involve either the frontal or the ethmoid sinuses. The mucocele of the maxillary sinus destroys the wall of the orbit and disturbs the eye. The author presents a case. A man, 59 years of age, showed a swelling of the lower lid and pressure on the right eye. These had existed for a number of months. The eyeball was deviated upward. At operation it was found that the protrusion was caused by a mucocele of the maxillary sinus which had perforated the orbital wall. The mucocele contained cloudy, yellowish-brown fluid of a tenacious character. Histologic examination of the wall of the cyst showed a chronic inflammation. It is believed that the process started with empyema confined to the antrum, with walls atrophied from pressure and that then the cystic protrusion developed.

A. KNAPP. [ARCH. OPHTH.]

Miscellaneous

RECENT ADVANCES IN THE TREATMENT OF CARCINOMA OF THE OESOPHAGUS FROM THE SURGICAL ASPECT. G. TURNER, J. Laryng. & Otol. **49**:297 (May) 1934.

Turner believes that the usual hopeless attitude in regard to cancer of the esophagus is not justified since the condition is not always rapidly fatal. There has been little attempt to deal with these growths radically at an early stage. Difficulty in swallowing has been allowed to drag on until it becomes not only a great discomfort but a serious menace to life. In many patients from three to six months have elapsed between the onset of symptoms and the stage at which some active treatment is seriously considered. There are frequent failures in attempted surgical excision of esophageal cancer, but the author describes one successful case.

The patient was a man, aged 58, whose difficulty in swallowing began only eight weeks before he was seen by the author. A preliminary gastrostomy was made, and the liver explored. About three weeks later the author performed the "pull through" operation under general anesthesia, which is divided into four steps: 1. The abdomen is reopened through a middle line incision carried high up between the left costal margin and the xyphisternum. The left lobe of the liver is detached from the diaphragm and turned to the right, exposing the abdominal portion of the esophagus. A quantity of 0.05 per cent solution of procaine hydrochloride is then injected around the lower thoracic portion of the esophagus, to distend the cellular spaces around it and to displace the pleura. The peritoneum over the esophagus is incised, and the enucleation is commenced by the forefinger introduced through the hiatus and worked up as far as possible around the tube. 2. A transverse or oblique incision is made first above the left clavicle, dividing the sternocleidomastoid muscle. The cervical portion of the esophagus is exposed by blunt dissection. The esophagus is then ligated and divided as low down as is feasible, but at least 2 inches (5 cm.) above the growth. 3. A return is made to the abdomen, and traction is exerted on the esophagus. When it is freed from its bed it is ligated at the cardia and cut away from the stomach, and the stump is securely buried by purse-string sutures. 4. The open esophageal tunnel is closed by suturing the left lobe of the liver over its mouth, and the abdomen is closed. This procedure was followed fairly closely, and the patient had a smooth preoperative course. Later a neo-esophagus was fashioned from a skin tube over the front of the chest and a loop of jejunum connecting this tube with the stomach.

In spite of many failures, Turner believes that radical surgical intervention holds some promise. It is better not to attempt the repair of the esophagus in situ, but after recovery to supply the defect by making a new esophagus by the anterior route. For a growth of the upper end he suggests an excision with formation of a cervical fistula and completion of a new esophagus. For a growth of the lower end the plan outlined is recommended, but for a tumor of the middle portion he believes that further consideration should be given to operation by the posterior mediastinal or transthoracic route.

EDITOR'S ABSTRACT.

ABSCESS OF THE BRAIN. K. W. MACKENZIE, J. Laryng. & Otol. **49**:357 (June) 1934.

Mackenzie reports in detail 8 cases of abscess of the brain. Six patients were cured—a recovery rate of 75 per cent. In 7 of the patients the abscess was the result of chronic suppurative otitis media. Four of these had abscesses of the temporosphenoid lobe and 2 of them recovered; 3 had cerebellar abscesses, and all of them recovered. In 6 of the 8 cases the abscess was actually drained at operation, and all the patients got well. In 1 of the remaining 2 cases, a decompression operation was performed, but before a final operation for drainage of the abscess could be carried out the abscess burst into the ventricle and the patient

died. In the other an exploratory operation was undertaken without success; ultimately the abscess burst through the surgical wound, and the patient died eight days later. These abscesses occurred during the years 1925 to 1933 inclusive, when there were 224 patients with chronic suppurative otitis media admitted to the ward for either a radical or a modified radical mastoidectomy. During the same period there were 481 cases of acute mastoiditis, but from none of these did an abscess of the brain arise.

It has been part of the author's technic to postpone operation until symptoms of pressure become well marked, by which time local immunity has developed. Delay also renders the discovery and drainage of the abscess easy and results in an immediate hernia of the brain on incision of the dura which, by compressing the dura against the bony margins of the trephine opening, lessens the risk of the onset of meningitis. In 7 of the cases reported drainage was established through a clean trephine wound of from 1 to 1½ inches (2.5 to 3.8 cm.) in diameter situated either in the squamous portion of the temporal bone or posterior to the sigmoid sinus. The eighth case of frontal lobe abscess was drained through the opening caused by osteomyelitis of the frontal bone. The author concludes that a "delayed" operation is necessary for success and that drainage through a large trephine wound is more likely to be successful than drainage through the mastoid alone.

EDITOR'S ABSTRACT.

RESULTS OF ENDONASAL TREATMENT OF PITUITARY TUMORS. M. HIRSCH, Ann. d'ocul. **170**:692 (Aug.) 1933.

Hirsch describes his transseptal method of operating on pituitary tumors and illustrates by means of photographs the various forms of these tumors. He used this method in operating on 233 patients and obtained remarkable results. He observed 57 of the patients for from four to thirteen and one-half years after operation and found that 41 showed improvement without relapses.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

LIMITATIONS OF ENDOBRONCHIAL THERAPY. H. SOLBRIG, Monatschr. f. Ohrenh. **68**:388 (April) 1934.

Solbrig discusses the causes of failure in bronchoscopic removal of foreign bodies. The use of a sound as guide sometimes pushes the foreign body farther away and makes it unextractable. Extreme peripheral localization is at times also a cause of failure. In another group of cases small light foreign bodies, usually multiple, may be aspirated into the smaller bronchioli which makes them unextractable. Localization of a foreign body in an upper lobe is fortunately rare, for its removal is difficult. Local changes in the lung, such as emphysema and empyema, may cause failure to localize properly or inability to remove a foreign body. In cases in which bronchoscopy fails, pneumonotomy must be considered. The greatest danger from pneumonotomy lies in cases in which secondary changes in the lung have occurred. It is therefore advisable to perform a pneumonotomy when indicated as early as possible.

EDITOR'S ABSTRACT.

ARGYROSIS OF THE NASAL MUCOSA. ALBANUS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:300 (March 12) 1934.

Albanus describes a case of argyrosis resulting from the ingestion of 9 Gm. of silver nitrate in solution in the course of three years. The skin was deeply impregnated. The mucosa of the nose and ear were less deeply impregnated, but more markedly involved in the regions reached by light than in those in relative darkness. Preparations of the skin and nasal mucosa showed a much greater deposit of silver in the skin than in the mucosa.

EDITOR'S ABSTRACT.

INTERNAL PNEUMOCEPHALUS FROM SEVERE SKULL FRACTURE. G. ENGELHARDT, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:426 (April 6) 1934.

Engelhardt describes the case of a man, aged 33, who sustained a fracture of the skull. The fracture line extended from the left outer orbital margin backward along the upper margin of the greater wing of the splenoid and ended in the middle of the squama of the temporal bone. The first roentgenogram was not taken until about twelve weeks after the accident causing the fracture. In this picture was seen a long, oval, light area, about 4 by 3 cm., which lay over the left frontal sinus and projected backward along the surface of the frontal bone. In addition two horn-shaped areas of lightness were seen which in their form, position and size corresponded to air-filled lateral ventricles. The source of this air is somewhat uncertain, but the author feels that the most likely entrance was by way of the opened frontal sinus. The air was gradually absorbed without active interference. Since operation was not necessary, lumbar puncture was not done. In certain cases, however, lumbar puncture is necessary for diagnosis or as a preliminary to operative intervention.

EDITOR'S ABSTRACT.

NEUROLOGIC DIAGNOSIS OF TUMORS OF THE POSTERIOR CRANIAL FOSSA. P. HILPERT, Ztschr. f. Hals-, Nasen- u. Ohrenh. **36**:3 (April 26) 1934.

The neurologic diagnosis of tumors of the posterior cranial fossa has been studied by Hilpert from several angles. The phylogenesis and ontogenesis of the cerebellum, as well as the projection of the spinal, olivary and pontine nuclei on the cerebellum and numerous observations of pathologic specimens, confirm the Edinger division of the cerebellum into a paleocerebellar and a neocerebellar section. A transverse partition is justified by the special relation of the lobus medius and the phylogenic and ontogenic significance of the primary sulci. The lowering of the neocerebellar hemisphere has a meaning only so far as the different parts are not equally provided with pontine fibers but are coordinated by nuclear regions of the pons. It is on this relationship that descriptions of the fiber connections of the human cerebellum must be based. The anatomic relations of the human auditory nerve and of the cerebellum are described in detail by the author.

The function of the cerebellum is fundamentally a unit. The physiologic function is the same throughout. It operates only under the influence of the cerebrum and in this way serves the higher coordination of the final movements. In this way purposeful and harmonizing movements are made, and with the help of intentional control, habitual individual movements are made automatic. The diagnosis of tumors of the posterior cranial fossa is hence difficult. As general symptoms of tumors of the cerebellum can be considered: headache, vomiting, usually increased pulse rate, choked disk and hydrocephalus. Under localizing symptoms the author lists two symptom complexes: dyskinesia and dystonia. Adiadokokinesis, disturbances of reaction to the finger-nose test and disturbance of speech thus are of some value in localizing tumors to the cerebellum. Of the dyskinetic symptoms, asynergy and asthenia are important. Of the numerous tonic symptoms of cerebellar disease, the two most important are past pointing and the deviation reaction. Static tremor in pure forms of cerebellar tumor is rarely observed. The pronation phenomenon and the frequent forced position of the head are of some value. Protrusion of one or both eyes, peripheral facial palsy, oculomotor disturbances and involvement of the pyramidal tract or lemniscus sometimes occur late in lesions of the cerebellum.

The diagnosis of tumor of the pontine angle is based on several localizing symptoms, so that wrong localization is rare. Three symptoms are of importance. These are crossed paralyses, conjugate paralysis of the eye muscles and trigeminal paralyses. The single reliable symptom of a tumor of the fourth ventricle is the vestibular seizure resulting from change of position. The cardinal symptom of a growth in the region of the corpora quadrigemina is the conjugate ocular palsy. In regard to the accessory methods of diagnosis, the author states that

the spinal fluid pressure in tumors of the posterior cranial fossa varies as does Queckenstedt's phenomenon. Occasionally tumor cells may be found in the fluid. In general, spinal puncture gives little aid in diagnosis. Ventriculography, on the other hand, is indispensable. Whether arteriography may have so many advantages over ventriculography that it can displace it must be decided in the future.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

ABSTRACT OF TRANSACTIONS BY ROBERT SONNENSCHEIN, M.D., CHICAGO

Sixty-Seventh Annual Meeting, April 6-7, 1934, Atlantic City, N. J.

JOHN R. PAGE, M.D., *President, in the Chair*

Morning Session, April 6

PRESIDENTIAL ADDRESS: BACTERIOLOGY OF ACUTE INFECTIONS OF THE MIDDLE EAR AND MASTOID. DR. JOHN R. PAGE, New York.

This article will be published in full in a later issue of the ARCHIVES.

FINANCIAL REPORT OF COMMITTEE ON OTOSCLEROSIS. DR. ARTHUR B. DUEL, New York.

Last year the income of the committee like that of all other organizations which have permanent invested funds, suffered. The income is between $20,000 and $25,000, and the fund for allocation was $15,500. To that was added $500 for immediate distribution, but allocations for the different fields of work had to be reduced. I am happy to say that despite the fact that less money was available for the work all the men engaged in research carried on in exactly the same way, and the actual scientific work on otosclerosis has not materially suffered from the decreased income. Perhaps the department which has suffered the most is the central bureau. There was material ready for a second edition of the current literature, but publication had to be suspended, and only that which has already been done was published. Through the activity of Dr. Fowler and his son the central bureau has provided a place where the Committee can gather to discuss the work. There is a permanent exhibit on the floor. Dr. D. Harold Walker replaced Dr. Crockett, and Dr. Rae succeeded Dr. McKernon, who resigned.

REPORT OF COMMITTEE ON OTOSCLEROSIS. DR. J. GORDON WILSON, Chicago.

In the past year the work of the Committee has covered a wide field, which may be briefly divided into two parts: (1) the anatomic work, including studies of embryology and pathologic anatomy, and (2) the physiologic work, including experimental investigations.

The first division has produced results which have markedly advanced the knowledge of the petrous temporal bone and of the diseases which attack it. The second division includes two definite lines of investigation: studies of (1) auditory problems capable of being studied in conditioned animals and (2) physiologic problems arising in connection with the investigation of the Wever and Bray phenomena. In this division pioneer work which has won international approval is being done.

Bast has devoted special study to the structure of the fissula ante fenestram. He found that though, as a rule, otosclerosis starts in the region of the fissula it generally attacks first not the tissue of the fissula (primitive intrachondral bone), but rather the surrounding bone. The otosclerotic patch may involve most of the capsule in the region of the fissula and obliterate the fissula near the middle ear, but the vestibular end of the fissula may remain unchanged and extend for a considerable way into the otosclerotic area.

Dr. Anson studied the fissula in 50 adults and made models illustrating the different types. A comparative anatomic study was made (1) of the formation, form, size and histologic characters of the stapes in the fetus, infant, child and

adult and (2) of the form and size of the stapes in adults, from exact measurements of 60 specimens. The structure of the intrachondral bone and the changes occurring in that bone with advancing age were also studied. It has been decided to make a further study of otosclerosis in relation to other osseous dystrophies. This is believed to be an important line of work. Dr. Edmund P. Fowler Jr. has been active in the study of otosclerosis and believes that the blue mantles hitherto regarded as a part of the otosclerotic process are really more an indication of a previous inflammatory reaction and occur in certain nonotosclerotic areas in which there is evidence of previous inflammatory reaction. He believes that his findings confirm the view of Dr. Edmund P. Fowler Sr. that there are signs of previous inflammatory reaction in all cases of otosclerosis.

Dr. Edmund P. Fowler Sr. has prepared 50 petrous bones of patients from whom audiograms (air and bone) were obtained during life. He has made a study of bone tissue cultures in vitro to observe the development of bone cells from fibroblasts. He has also studied the petrous bones of congenitally deaf dogs and cats from Dr. Duel's collection in order to investigate the mendelian factors in hereditary deafness. He has studied the action of quinine (when given to pregnant women) on the fetus and on the infant.

Dr. Culler has investigated the problem of the round window. He found no evidence that blocking the round window or immobilizing the membrane improves hearing. From his experiments he believes that the improvement in hearing is a phenomenon due not to the eighth nerve but to conduction through highly ionized body fluids in bone and tissue. When a good conductor, such as a saline solution, is used to connect the membrane of the round window with the bone or membrane of the promontory the pick-up from any nearby bone or tissue is increased.

He also found that when the whole auditory tract, from the peripheral nerve to the cortical area, is irradiated with roentgen rays of which the voltage, amperage, wavelength, duration and approximate energy directed to a given point in the cranium are known, a small dose of soft (90 kilovolt) rays is followed in every case by a rise of 5 decibels. The change appears about ten days after the irradiation, suggesting that it is due to some metabolic change in the nerve tissue. It passes away in three or four weeks, and the animal reverts to its former hearing capacity. Dr. Culler found that exposure of an animal to loud tone (1,000 cycles) for several days lowered the animal's hearing capacity by 40 decibels. Interrupted sound was more effective than continuous sound in this respect. Dr. Culler is at present investigating the relative value of the several auditory pathways as contributors to actual hearing. He found that complete elimination of one cortex impaired the actual hearing to a very slight extent (5 decibels).

Professor Babkin and Professor Collip have studied the effect of parathyroid extract and of viosterol on the activity of the cerebral cortex and on auditory capacity in dogs. These drugs cause depletion of calcium, and disturbed calcium metabolism appeared to affect the activity of the cerebral cortex. Abnormal calcium metabolism may, therefore, affect the cerebral centers for hearing. Investigation into the phenomena of chronic alcoholism in dogs showed that it had no effect on the hearing capacity. Operations to produce cerebral anemia of the temporal lobe of the hemisphere (auditory zone) resulted in lowering the upper limits of hearing in dogs. The balance between excitation and inhibition was upset, and the animal became extremely cowardly.

The department of physiology of Harvard University reports the work of Dr. Davis and his colleagues. They have found that when sound waves are transmitted to the ear two distinct electrical phenomena are produced: (1) the electrical "microphonic" response arising in the cochlea, (the cochlear or Wever and Bray effect) and (2) the true auditory action currents passing up the eighth nerve. The cochlear response is not limited to the auditory tract and persists, although reduced, for many minutes after death. The action currents correspond to action currents in sensory nerves and transmit vibrations up to 1,000 double vibrations. The true action current is localized in the auditory tract and disappears instantly at death. The auditory mechanism is, therefore, dual in character, the mechanism for high pitches differing radically from that for low pitches.

OTOSCLEROSIS IN A SEVEN MONTH HUMAN FETUS (DEMONSTRATION OF SLIDES). DR. LOUIS K. GUGGENHEIM, St. Louis.

All otolaryngologists are familiar with the microscopic picture of otosclerosis: (1) the sharp line of demarcation between the focus and the capsule, (2) the prepared or degenerated capsule, (3) the fibrogenic marrow spaces containing engorged vessels and surrounded by blue-staining calcified connective tissue, (4) the blue mantles, (5) the areas of resorption and (6) the osteoclasts (sometimes present, often absent). The first groups of slides represent the second and common phase of otosclerosis. Physicians are probably less familiar with the third stage, in which condensation has occurred, the bone stains red, the vessels in the marrow spaces are smaller and fewer and the marrow spaces, now fibrous rather than cellular, are small and few.

Concerning the earliest phase (stage 1) of otosclerosis, one has had nothing but speculation. It has generally been believed that resorption occurs and that fibrogenic marrow creeps into the hole left by the resorption and forms the focus.

In June 1933 while examining serial sections from a 7 month fetus I noted a 1.5 mm. focus of blue-staining bone in the inferior wall of the round window niche. In order to determine whether or not this was normal or pathologic, sections from 16 fetuses of the same age were examined: 2 at the department for outpatients of the McWilliam Hospital and Oscar Johnson Institute, 5 through the courtesy of Dr. E. P. Fowler Jr. at the Medical Center, New York, and 9 through the courtesy of Dr. Jones and Dr. Hemsath at the New York Post-Graduate Medical School and Hospital. Nothing corresponding to the blue focus was found in any of the 16 fetuses examined. An attempt was then made to fit the picture into the twenty pathologic conditions of the capsule as described by Nager. While the focus was similar to the changes in Paget's and in Recklinghausen's disease the focal nature excluded these two conditions. The details of the focus are: (1) short-fibered, blue-staining web bone, (2) absence of the normal hematopoietic-osteogenic marrow and the presence of fibrogenic marrow with cells strikingly similar to those found in the second stage of known otosclerosis, (3) degeneration of osteocytes and bone matrix (loss of calcium in the red bone), (4) degeneration of cartilage rests and (5) invasion of the red web bone and cartilage islands by fibroblasts and blue bone.

The conclusions reached are as follows: Otosclerosis must have an earlier pathogenic phase than any heretofore seen. Five distinct features of the condition presented by the 7 month fetus correspond to the recognized picture of otosclerosis. The picture is unlike anything I have found in normal fetuses and is to be regarded as the first phase of otosclerosis until proved to be either a normal deviation or another pathologic process.

DISCUSSION

DR. EDMUND P. FOWLER, New York: It is only in the embryo that one can determine whether any structure has a phylogenetic relation with another. Dr. Guggenheim has sought this information, and he contends that the foot plate of the stapes is developed from the same anlage as the labyrinthine capsule, instead of from Reichert's cartilage, as hitherto reported. If his contention is true it will show how disease of the anterior border of the capsule may involve the annular ligament and the foot plate of the stapes. I cannot discuss the embryologic aspect of this report adequately, but a study of the specimens has shown that the endochondral capsule has a bizarre structure, consisting of strands of parallel fiber bone, intertwining about islands of basophilic cartilage. New bone growth is of short web fiber type and contrasts sharply with more normal bony structure. There is a tendency, therefore, to give the name otosclerosis all short web fiber bone in the labyrinthine capsule. My associates and I have in our collection 2 cases showing an invasion of the labyrinthine wall from within. In both cases short web fiber bone is being laid down. However, we doubt if this direct invasion can be the true mechanism of otosclerosis.

Dr. Guggenheim's argument is that blue-staining bone is pathologic bone. It may be pathologic or not, but it need not be otosclerotic. It does not occur at

the sites of predilection of otosclerosis. It is below the round window and quite deep in the bone, whereas otosclerosis tends to occur in the niche of the round window.

From studies I am inclined to believe that there must be some local lesion which is the inciting factor of the otosclerotic process. The young seldom show otosclerosis, the fetus never (this case being the exception). The percentage of otosclerosis and of frank or masked otitis media advances with age.

DR. LEROY M. POLVOGT, Baltimore: I want to congratulate Dr. Guggenheim on his keen interest in the study and advancement of the knowledge of otosclerosis. His work is a great inspiration.

When I was asked to discuss this paper I first read the preliminary report of this case published in the ARCHIVES OF OTOLARYNGOLOGY. My next step was to study the temporal bones of 75 fetuses. It is sometimes difficult to point out otosclerotic foci with black and white lantern slides. Therefore, I asked to study Dr. Guggenheim's original sections. Having carefully studied the sections and having seen the lantern slides, I am unable to see any pathologic process in the case presented. I believe that the bone in this case is normal for a fetus of 7 months.

DR. T. H. BAST, Madison, Wis.: It is hard to tell from a black and white slide what the condition is. I have sectioned from 75 to 100 fetal ears and have never seen one variation that I should be willing to call otosclerosis.

DR. FRANK R. SPENCER, Boulder, Colo.: Did the mother of the fetus have a diet that was deficient in vitamins or in calcium? Dietary deficiency in the mother would play an important part in early otosclerosis.

DR. LOUIS K. GUGGENHEIM, St. Louis: This discussion has been frank, albeit somewhat inconclusive. I have no family history in this case. Studies on a 2 month fetus, (with a mother with classic otosclerosis) are now being made. The fact that blue-staining bone is seen in healing fractures, etc., and also in otosclerosis, is no argument against its occurring in otosclerosis. I pointed out numerous areas of resorption. The cement line is not always present and is hard to show. This material is from only a 7 month fetus, in which the cement line could not exist because complete resorption of the capsule has not occurred. I consider that the focus is located almost at the classic site reported by Panse. As to web bone, normal bone is formed by osteoblasts in rows, while otosclerotic bone is a differentiation from mesenchyme and fibroblasts. I believe that this specimen presents a focus which does not fit in with normal embryology. I have not seen such an appearance in a normal fetus, but it may be a normal variation.

STUDIES OF OTOSCLEROSIS BY MEANS OF ULTRAVIOLET RADIATION. DR. EDMUND P. FOWLER JR., New York.

Observation of photomicrographs by means of ultraviolet radiation brings out the fiber structure of bone in unstained sections. It shows that all kinds of fiber structure can be found in otosclerotic foci. However, the most common type of fiber structure is the short fiber or reticular type. In old foci this may be partially replaced by lamellar or "shell bone." Rarely, small areas of parallel fiber bone are seen. Studies of otosclerosis by means of ultraviolet radiation indicate that otosclerosis is a formation of new bone rather than a change in old bone. Its short fiber web structure is characteristic of regrowth. Similar bone is found from the femur to the labyrinthine capsule in embryos. It is also found in osteitis in adults, in the renal capsule of rabbits in which the vessels to the kidneys have been tied experimentally, and in healing fractures. It stands out sharply in the anterior border of the oval window because the normal bony structure at that point is very peculiar, and it can cause clinical symptoms there.

Areas of new growth of short fiber web bone can be demonstrated throughout the labyrinthine capsule in cases of known or masked osteitis either with or without otosclerosis. This suggests that inflammation may well be of primary impor-

tance in the production of otosclerosis. Because new bone of this type has been produced by venous stasis the rôle of inflammation is probably that of thrombosis of the vessels to the areas involved rather than that of direct invasion of the wall.

DISCUSSION

DR. T. H. BAST, Madison, Wis.: Dr. Fowler mentioned short web bone. One can get all grades between the short web type and the long type. It all depends on the speed with which bone is laid down. Dr. Fowler regards blue-staining bone as pathologic. It has been shown in many bones that are normal. I believe that remnants of cartilage which are really intrachondral bone are very resistant, while ordinary endochondral bone is less resistant. These islands of cartilage project into the osteoporotic areas. The islands are excavated later.

DR. STACY R. GUILD, Baltimore: I do not believe that enough evidence is available as yet to say one thing or the other. A few cases prove nothing definite.

DR. J. GORDON WILSON, Chicago: Additions to the knowledge of otosclerosis are sure to follow the use of a new technic or of a new instrument. Dr. Fowler's report is a good example. The photomicrographs are excellent and are convincing for many of the points he makes, for instance, the sharp line of demarcation between the otosclerotic focus and the normal capsule and the irregular bone in the focus, partly lamellar and partly primitive bone. Though this has been observed before, so far as I know it has never been seen so clearly.

His study is directed to the conclusion that otosclerosis is a local process, dependent on a vascular change in the course of known or marked otitis media in a predisposed person. This vascular change is a change in the vessels themselves, stasis, thrombosis, etc. This is not a new hypothesis, and partly to test it I asked Dr. Dworik to remove the sympathetic chain in the neck of a conditioned cat. The results of such a removal are dilatation and stasis in the vessels of the head; so far, no effect on hearing has been noted. The animals are still living, but a postmortem examination will be made and reported on. This does not invalidate Dr. Fowler's argument; neither does it substantiate it. Along this line more exact methods of attack suggest themselves. Another argument against this hypothesis, i. e., that there are no signs of inflammatory reaction, has not been satisfactorily explained.

I agree with the anatomic observations. The fibers of the capsule never run into the focus, and the line of demarcation where the focus joins the periosteal bone is not marked. The interlacing *geflechtartig* bone in the capsule could with advantage receive an English name. Why not call it "primitive bone," since, as Bast has shown, it is similar to primary bone laid down elsewhere?

I wonder whether too much stress is not being laid on the unique anatomic character of the capsule and otosclerotic focus and too little on its unique position. Bone is not a permanent structure; it is subject to constant change, decidedly so in the petrous temporal region. The capsule which protects the membranous labyrinth does not increase materially after birth. It maintains unaltered in its center a fully developed structure. Yet this capsule is surrounded by bone with considerable peripheral periosteal growth in all its dimensions and subject to changes of position in keeping with the development of the skull. This appears to me to be the significance of this unique capsular bone with its islands of cartilage, its irregular weblike fibers and its few lamellae. In this ossified capsule there is a structure firmer and more resistant to pressure than cartilage, yet more plastic than lamellar bone.

DR. EDMUND P. FOWLER JR., New York: Dr. Wilson mentioned that there was no demarcation between otosclerosis and certain parts of periosteal bone. Periosteal bone and sclerotic bone look much alike, and therefore the line of demarcation is hard to find. In regard to cases of Paget's disease showing more osteoclasts than cases of otosclerosis, osteoclasts occur only where active destruction and growth of bone are going on. In highly active otosclerotic processes one also finds osteoclasts.

I agree with Dr. Bast that too much emphasis has been laid on considering short fiber bone pathologic. Normal parallel bone of the endochondral capsule is often as primitive as the short fiber web bone of the embryonal capsule.

INFECTIONS OF THE TEMPORAL BONE WITH SECONDARY MANIFESTATIONS. DR. MARVIN F. JONES, New York.

Three routes for meningeal invasion from the middle ear are demonstrated in the pathologic preparations: (1) directly through the temporal bone, (2) through the bone canals housing the nerves and (3) by traumatic dislocation of the stapes.

The transmission of infection through the temporal bone may be facilitated when an acute infection is added to a chronic degenerative process.

The membrana tympani is a comparatively resistant structure, peculiarly so following the changes produced by prolonged inflammation.

Resolution of the membrana tympani may be present concurrently with a destructive process in the mastoid antrum of an infant. No convincing evidence was found that the infected mastoid process acted as a focus for general infection.

The classic symptoms of a parenteral infection in infants occurring concurrently with mastoiditis improve following mastoidectomy. The earlier the operation is performed after gastro-intestinal symptoms are manifest, the lower the mortality. The premature closing of the mastoid incision may cause a recurrence of gastro-intestinal symptoms.

The pathologic changes observed in the temporal bones examined do not represent a terminal pathologic process but give histologic evidence of chronicity.

The observation of intralabyrinthine pathologic changes suggests the theory that the vomiting observed, particularly in cases of meningitis, may be the result of labyrinthine irritation.

DISCUSSION

DR. L. W. DEAN, St. Louis: There are many important general clinical conditions associated with infection of the ear; among these are: meningitis, abscess of the brain, septicemia, cardiac lesions, nephrosis, nephritis, malnutrition, cervical adenitis, diarrhea in infants with dehydration, bacillary diarrhea, fever, cyclic vomiting, chorea, arthritis, pyelitis, chronic pulmonary infections, diabetes and neuritis of the gasserian ganglion.

Infection of the ear can play a prominent part in the clinical picture in any of these conditions and must be eradicated. In hemorrhagic nephritis associated with acute otitis in children, the urine can be cleared of blood by operation on the ear and by the stamping out of infection. Tonsillar infection frequently causes hemorrhagic nephritis, and so does infected lymphoid tissue in the pharynx. A coexistent aural infection has a marked influence on diabetes. In chorea, cardiac disease and rheumatic fever in children it is highly important to eradicate infection about the ear, nose and throat. However, the aural infection is not the only cause of the systemic condition in these children. Their fever has to be controlled by salicylates, and it often takes two years of the best nutritional and hygienic treatment to get them back to normal weight and strength.

Lobar pneumonia often occurs with acute otitis media; both are secondary to an infection in the upper respiratory tract. Acute otitis influences diarrhea in infants, and while it is not a direct causative factor, it creates an unfavorable element, and the child is greatly helped by eradication of the otitis. Such infants suffer from malnutrition before otitis or diarrhea develops, and they show a decreased acidity of the gastric juice. This permits organisms from the lower part of the large intestine to permeate the small intestine, causing diarrhea. Consequently, errors of diet, whatever they may be, must be corrected in order to get the best results in the treatment of infection of the ear.

DR. F. A. HEMSATH, New York: My interest in this report has been from the pathologic and technical side. It is important to correlate the laboratory observations with the clinical material.

DR. JOHN B. RAE, New York: Unfortunately, these cases are rarely found in special hospitals, where only patients with frank aural conditions seek admission. They occur in the pediatric services of general hospitals, and the moral of the story seems to call for complete cooperation between the pediatrician and the otologist. When the former has reached the end of his rope in treatment the latter should have the deciding vote about operation, and this decision should be reached at a date sufficiently early to avoid the possibility of a fatal outcome.

DR. D. E. S. WISHART, Toronto, Canada: These facts emphasize the difficulty and the importance of the diagnosis of mastoiditis in young children.

An infant is going downhill; its ears are discharging; there are no external signs of mastoiditis. Is the cause of the downward progress an unsuspected mastoiditis? That is the problem in a nutshell.

Consultants, as a rule, are practicing otologists familiar with the picture of mastoiditis in the adult and inclined to doubt the existence of mastoid infection without external signs. They often have to be led to the operating room and to be shown that the pathologic condition is really in the mastoid.

Dr. Jones has shown that in nearly all the cases he investigated he has found a pathologic condition worthy of surgical intervention, but it would be a gross error to suppose that every infant requires an operation on the mastoid, because many infants with the clinical problem have entirely healthy temporal bones. It is a fact that the percentage of infants who die of acute otitis media is strikingly high, yet many die of intestinal symptoms without having had otitis media.

DR. W. J. MCNALLY, Montreal, Canada: In the group of cases described, in which the child is going downhill and the mastoid may be a factor without any of the usual signs of mastoiditis, what are the signs by which one can be guided in diagnosing a mastoid condition?

DR. MARVIN F. JONES, New York: I wish that Dr. McNally's question were an easy one to answer. There is a cooperative group of pediatricians, and they have been "sold on" the idea of aural examination. All these cases have been thoroughly studied by both pediatricians and otologists before conclusions were reached. The errors have been in omission rather than in commission. No mastoid has been opened without a good cause. The cases studied were not selected cases. They were 9 cases which occurred in children under a specified age and in which a routine autopsy was performed. The point which Dr. Wishart brought out as to the spread of the gospel of group diagnosis is important. While it is true that one cannot operate on the mastoid when the child has diarrhea but has a normal drum, such an operation is very slight; it is performed under local anesthesia, with incision of the periosteum and with the minimal amount of shock to the patient. If the cells are there they have to be evacuated. As to criteria for diagnosis, I consider that in doubtful cases an exploratory antrotomy is justifiable. It is difficult to tell the difference between a resolving drum and one housing an involvement of the mastoid. The chief consideration is the condition of the child. If it is going downhill one is not justified in withholding the operation. In our service my colleagues and I had 45 cases associated with gastrointestinal disturbance and 11 deaths. That mortality is high, but we accept practically moribund patients.

Afternoon Session, April 6

THE ELECTRICAL ACTIVITY OF THE COCHLEA IN CERTAIN PATHOLOGIC CONDITIONS. DR. HALLOWELL DAVIS and DR. MOSES H. LURIE, Boston.

In our experimental work on the physiology of the ear we accumulated a small group of animals, cats and guinea-pigs with various abnormalities of otologic interest. All the animals which in the experiments gave indication of functional abnormalities of any kind were examined post mortem and studied for pathologic changes. The animals fall into two groups: (1) those animals with infections of the middle ear, (2) those with normal middle ears.

Group 1.—In the group with infection of the middle ear were 2 cats and 1 waltzing guinea-pig. All these animals had a marked infection of the middle ear and pus and granulations filling the bulla, and the stapes was the only ossicle found.

CAT 1.—The electrical (cochlear) response and the action potentials from the eighth nerve were both obtained but with elevated thresholds of 50 S.U. The microscopic examination showed that a passive congestion of the cochlea but no infection was present.

CAT 2.—This animal gave a response from the cochlea which was essentially normal except for an elevated threshold of about 40 S.U. No action potentials could be obtained from the eighth nerve or the midbrain. The histologic examination showed a marked infection of the middle ear with infiltration of the bony capsule. The organ of Corti was normal in appearance. The spiral ganglion showed a diminution and degeneration of ganglion cells. The cochlear nerve showed degeneration and a chronic infection of the sheath, especially where it was in contact with the modiolus.

GUINEA-PIG.—The waltzing guinea-pig gave no cochlear or auditory nerve response, and the histologic picture was that of an infected cochlea with pus present in the scalae and throughout the cochlea.

Group 2.—In the group with normal middle ears there were 3 cats and 2 waltzing guinea-pigs.

CAT 3.—This was a white cat with the right eye blue and the left eye yellow. The right ear gave no electrical response. The left ear was normal in all respects. Histologically the right ear gave the following evidence: The organ of Corti was completely degenerated. Reissner's membrane and the basilar membrane were fused. The spiral ganglion was degenerated; practically no ganglion cells were present. The cochlear nerve showed a marked diminution of fibers. In the left ear the cochlea and cochlear nerve were normal.

CAT 4.—This animal gave a cochlear response (picked up from the round window) only when strong stimulation was used. There was a loss of hearing of from 40 to 100 S.U. The response was absent at frequencies about 4,096 cycles per second. The curve of the audiogram showed a definite fall beginning at about 350. The histologic examination showed an absence of the organ of Corti in the basal whorl, with the organ of Corti appearing normal for the upper one and one-half turns.

CAT 5.—This was a castrated white cat with blue eyes and pink eyegrounds, which acted as if it was deaf.

On the right side the cochlear response was within normal limits. The response from the auditory nerve showed a loss of about 40 S.U. beyond that of the cochlear response, physiologically giving a picture of a normal organ of Corti and abnormality of the neural mechanism. The microscopic examination showed a hemorrhage in the cochlear nerve.

The left ear showed a loss of sensation of from about 35 to 60 S.U. for the cochlear response and auditory nerve potentials, especially for the middle range. The microscopic examination showed an infected inner wall of the middle ear with the stapes filled and covered with thickened periosteum.

GUINEA-PIG 1.—This waltzing guinea-pig gave no response to stimulation. The histologic examination gave the following evidence: The organ of Corti was definitely degenerated for at least two and one-half turns from the basal end, the spiral ganglion was degenerated throughout, two upper whorls showing the organ of Corti present but with degenerative changes.

GUINEA-PIG 2.—In the second waltzing guinea-pig the right ear gave response to tones up to 1,800 vibrations per minute with a loss of from 75 to 100 S.U. The left ear showed a similar picture with loss of from about 65 to 80 S.U. The histologic evidence for the right ear was a complete degeneration of the organ of Corti for the two and one-half basal turns. The spiral ganglion also showed marked degeneration. The upper one and one-half turns showed an apparently

normal organ of Corti with a normal-appearing spiral ganglion. The left ear gave a similar picture except that there was complete degeneration only in the two basal whorls.

Conclusions.—These observations on animals showing pathologic changes, combined with previous observations on normal animals, confirm the views that:

1. The cochlear response depends on the organ of Corti, for (*a*) it is absent when the organ of Corti is absent; (*b*) it has never been absent when the organ of Corti has been entirely normal, and (*c*) partial degenerations or deficiencies of the organ of Corti give partial, and sometimes complete, deficiencies in the cochlear response.

2. Nerve impulses are probably initiated by the cochlear response, for (*a*) the threshold curves for both run parallel in most animals; (*b*) nerve impulses may be seriously deficient or absent with the cochlear response present, but (*c*) nerve impulses have not been found in the absence of the cochlear response, except (1) when the threshold of the latter is raised by unfavorable local electrical conditions of detection or by interference from the nervous response and (2) in one doubtful, incomplete case.

3. The basal portion of the cochlea responds to high tones and the apical portion to low tones, but with a rather wide extent of physical vibration to strong tones. Deficiency of the organ of Corti in the basal turn causes a greater elevation of threshold in the high than in the low tonal range, but no abrupt transitions have been encountered.

4. The cochlear response is probably a good indicator of the activity of the organ of Corti, but the extent of an animal's hearing can be better evaluated from the action potentials of the auditory nerve. Even this may lead to error in cases of true central deafness.

A Modification of Auditory Theory. Dr. Moses H. Lurie, Dr. Hallowell Davis and Mr. A. J. Derbyshire, Boston.

This article was published in full in the September 1934 issue of the Archives, p. 390.

DISCUSSION

Dr. Stacy R. Guild, Baltimore: So many interesting points have been presented by the speakers that these two papers are more like a whole group of reports; to discuss them properly would take as long as to present them. I congratulate the authors on the extreme sensitivity of the apparatus they are using; to be able to obtain such graphic records from the nerve at acoustic intensities comparable to the thresholds of human auditory acuity provides a basic tool that opens up additional important fields to experimental observations.

While I am not in a position to question the actual observations on masking, I question the interpretation that the so-called masking effect is primarily produced in the peripheral end-organ of hearing rather than as a result of cortical "confusion." The difference that "paying attention" makes in the hearing of the elements of a complex sound indicates that all the parts of the sound reach the cortex.

Dr. C. C. Bunch, St. Louis: The authors lead one to believe that animals can be used for experimental work in auditory acuity, and that observations on histologic preparations can be correlated with the results secured from auditory tests. If this is the case experiments can be shorter and more economical. In human subjects there are so many varied factors that such correlation becomes tedious and expensive.

The illustration showing the depression in the wave form while records of the action currents are being made with the ear receiving a stimulus points to some sort of interference which is interpreted as a "line busy" effect. This is contrary to the usual conception of monaural hearing. It is true that in binaural hearing sound may enter both ears and be perceived only in one. Stewart and others

have demonstrated these effects in their study of sound localization. Otologists, however, confine their functional hearing tests to investigations concerning the pitch and intensity of tones.

DR. WALTER HUGHSON, Baltimore: I agree with Dr. Guild as to the accuracy of the findings and the finesse displayed in the performance of the experiments. In Baltimore otolaryngologists are more concerned with the end-organ itself than with the neurophysiology of auditory pathways. The method used there for detecting nerve impulses may be somewhat cruder than the procedure just described, and the results represent a combination of the cochlear effect and action current. In all of the experiments one ear is used as a control and the other for experimental purposes. Usually identical responses are obtained from both ears, provided they are normal. Occasionally, however, discrepancies appear in the two ears for any given frequency. I am interested in Dr. Davis' explanation of the damping and enhancing effect which the two currents may have on one another, depending on whether they are in or out of phase at any given moment. This would more readily explain the slight difference which is noted occasionally between the two ears than the theory of experimental error. Several years ago Dr. Crowe and I suggested that if an ear failed to transmit any tone or series of tones this was due to pathologic changes either in the end-organ or in the auditory pathway itself. Any such failure to transmit has been explained after sections of the ear were made. Dr. Lurie's cases bear out this point. In a large series of cases there have been many animals with infections of the middle ear or osteomyelitis of the cochlear wall. There have been many with infections following operation, several with infectious or chemical labyrinthitis, spontaneous lesions of the nerves, and one with an actual bony ankylosis of the foot plate of the stapes—besides the 16 cases in albino rats reported by Howe. In every instance these lesions have either decreased the intensity of transmission or eliminated it.

DR. JOHN GUTTMAN, New York: I have come to the conclusion that sound produces four effects: (1) a reaction on the sense of hearing, (2) a reaction on the nerve impulse, (3) electrical disturbance in the cochlea and (4) electrical disturbance in the nerve. Wever and Bray believe that the electrical disturbance in the nerve is the main disturbance. Davis thinks that the cochlear spread goes over from the cochlear disturbance to the nervous disturbance, or the reverse. However, when I cut the nerve there was no change in the disturbance from the cochlear. Thus, I thought that there were some nerve elements in the cochlear ganglion and that perhaps these elements were responsible for the local disturbance in the cochlea. I have also produced atrophy of the ganglions, and yet the cochlear response remained the same so far as I could judge. I have concluded that there are two types of electrical disturbance in the ear; one has no refractory period and goes up to 8,000 frequencies per second, whereas the action current stops at 1,000 per second. I think that it is unique to find an organ with two types of responses. I believe that there is a relation between the disturbance in the cochlea and that in the nerve, but I think that the cochlear disturbance influences the nerve, whereas a lesion in the cochlea can abolish the impulse from the nerve as well as that from the cochlea.

DR. T. H. BAST, Madison, Wis.: In working with the Wever and Bray method Dr. Eyster and I have come to the conclusion that what we picked up was electrical spread from the cochlea and not action currents. At the meeting in Cincinnati Dr. Davis demonstrated the cochlear spread, but he also demonstrated action currents picked up from the brachium of the inferior colliculus. Dr. Eyster was convinced of this, but I was somewhat skeptical. We therefore decided to check on these experiments. According to Davis, deep anesthesia blocked these currents, but we did not obtain uniform results with this method. We decided that the crucial test would be to cut the eighth nerve while the impulse was being picked up from the contralateral brachium of the inferior colliculus. If the pick-up is true action current, cutting the eighth nerve should cause it to be lost. When the nerve was cut the volume dropped markedly but the current

was not abolished. This left the possibility that the sound entered the homolateral ear and reached the brachium of the inferior colliculus through the few uncrossed fibers. We then cut the other eighth nerve, and the reception was entirely lost. The electrical spread, however, could still be picked up from the cut end of the eighth nerve. We believe, therefore, that Davis and Lurie have really demonstrated action currents, for which they should be congratulated.

I should like to ask Dr. Davis whether the cathode ray oscillograph wave picked up from the round window was the same as that picked up from other regions around the ear, for instance, from the tentorium?

Our observations have agreed with those reported, but the interpretation of the mechanism of hearing is not easy. The physicians from the Johns Hopkins Hospital believe that the organ of Corti is the only source of these initiating impulses. I do not think that all the facts are known as yet, and the possibility of the influence of Reissner's membrane should not be overlooked. Potentials can be set up in a permeable membrane with fluids on both sides. This is known as endosmosis. A disturbance of the fluid starts a flow of potential. Reissner's membrane is a suitable structure, and the disturbance by sound waves may release a flow of potential. This released energy may be picked up by the organ of Corti and transmitted to the brain as action currents. Dr. Eyster produced this phenomenon with an artificial membrane and cell. One picture presented by Dr. Lurie seemed to substantiate this theory. In that case the cells of the organ of Corti and those of the spiral ganglion were intact, yet conduction was poor. Reissner's membrane was collapsed, and this may account for the poor conduction.

Dr. M. Josephson, New York: In regard to the remarks of Dr. Bast, I wish to state that I made some studies on the transmission of sound through capillary tubes of approximately the same dimensions as the scalae and found it impossible to transmit anything less than maximal intensities. Therefore, it appears impossible that the cochlear spread potentials originate in the organ of Corti, for if they did it would be impossible to explain the response of the cochlea to threshold stimulation, because tubes the size of the scalae do not transmit sound as sound. It is more probable that currents of cochlear spread originate at the portal of entry of sound into the cochlea and that sound is conveyed through the scalae as electrical audiofrequency; in other words, the direct stimulus of the end-organ is electrical and the cochlear spread is potential, not sound as such. The only other explanation is the original idea of Helmholtz, that the promontory and the cochlea respond to stimulation by sound as a whole, rather than to its individual parts.

Dr. R. Lorente de Nó, St. Louis: I think that the Boston men have been telling me my own idea. This mechanism could explain the electrical response of the cochlea, but it is not proved that the electrical disturbance is produced in the hair cells of the organ of Corti. I am working on the following experiment: If one takes the cochlea with the organ of Corti and cuts the cochlear nerve at the entrance to the scala media; the cells do not degenerate at first, but later they all degenerate. Then the hair cells lose their differentiation. If after a certain time the electrical potential is still there one can assume that the response is in the organ of Corti, but this needs to be proved. Dr. Davis seems to advocate the localization of pitch not by the frequency of the impulse or by the number of active fibers but by the type of fibers which are conducting the impulse. This is similar to the theory of Helmholtz, but if some parts of the basilar membrane respond to pitch there must be independent stimuli going to the cochlea from the cerebral cortex. Such paths have not been demonstrated. Perhaps excitation of the cochlea carries a local sign at the same time that it produces another phenomenon. According to this theory there could be various parts with independent paths to the cortex.

Dr. Moses H. Lurie, Boston: I do not agree with Dr. Bast's contention that Reissner's membrane may be the cause of cochlear spread. In 1 of the waltzing guinea-pigs observed, Reissner's membrane was normal in position and appearance.

yet no cochlear response could be obtained. In this animal there was a definite change in the organ of Corti and in the spiral ganglion. If Reissner's membrane was the cause, it should have been possible to obtain cochlear spread over the entire range. As reported, in several of the animals there was a definite limitation of the cochlear response which correlated with the presence of the organ of Corti. I do not place much emphasis on the position of Reissner's membrane unless it is fused with the basilar membrane. Dr. Lorente de Nó suggested the production of degeneration of the organ of Corti by operative measures on the cochlear nucleus. My associates and I are planning to perform this experiment in the near future. It is a question of getting animals young enough (we believe) to have any success with this type of degeneration.

Dr. Hallowell Davis, New York: The experiment of damaging the cochlear nucleus, which Dr. Lurie mentions, was suggested by Dr. Lorente de Nó during his visit to the laboratory.

In answer to Dr. Guild, I do not deny the possibility of central masking, but it is certainly true that some masking, at least, is peripheral. One mechanism has been described. Another possibility is that purely physical interference of sound waves reduces the vibrations of the basilar membrane.

The parallelism between the cochlear response and the action potentials of the nerve certainly tends to justify the type of experimental procedure employed by Dr. Hughson. There are many experiments in which a mixture of the two responses is of no disadvantage, particularly in studying the mechanism of transmission in the middle ear. In other situations interpretation may become extremely difficult. I admit freely that in my own experiments I have modified the situation by opening the bulla. I believe that the effect is a minor one, but this must be checked experimentally.

Dr. Lorente de Nó was not the only one to make his interpretation of the origin of cochlear response. It was also my own original interpretation. I am now convinced of the important part played by the organ of Corti, but at first I believed precisely the opposite. My present view is a working hypothesis with some fairly good evidence in its favor. There may be endosmotic and other potentials in addition. The theory which I prefer is one which regards the sensory cell as analogous to a piezo-electric crystal the potentials of which are altered by the stresses caused by movements of the membrane. This coincides with the view advanced by Dr. Guttman.

In regard to independent pathways in the central nervous system, it is comforting to know that an anatomic basis for them exists, for one needs them theoretically. The nerve impulses of rather high frequencies detected in the auditory nerve certainly do not reach the cortex. At the level of the medial geniculate ganglion only the lowest frequencies can be recognized. A frequency theory of pitch is, therefore, impossible for high tones. By using a series of clicks at 64 per second, one can recover this frequency from the auditory cortex, but on listening to this stimulus one hears a buzzing which is not a pure tone. Therefore, for these low frequencies, as well as for high ones, pitch cannot be determined by the frequency of impulses but must depend on which portion of the cortex is activated. An attempt has been made to find a "map" of the organ of Corti on the cortex, that is, to locate particular regions activated by particular tones, but this attempt has been unsuccessful. The only definite cortical responses which can be identified are an "on-effect" and an "off-effect." These are present throughout the auditory cortex and are merely signals of activity which give no clue as to the localization which the theory demands, and which Dr. de Nó states is possible on anatomic grounds.

Ménière's Disease: Symptoms, Objective Findings and Treatment in Forty-Two Cases. Dr. Walter E. Dandy, Baltimore.

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MÉNIÈRE'S SYMPTOM COMPLEX: MEDICAL TREATMENT. DR. A. C. FURSTENBERG, Ann Arbor, Mich.

The chief characteristic of Ménière's disease is a violent attack of vertigo in a patient with deafness, usually impaired cochlear function on both sides, but more profound in the ear to which the symptoms are referred, nausea, vomiting and spontaneous nystagmus in the severe seizures and persistent tinnitus (often bilateral), increased during the paroxysm. Vestibular tests are of little significance. At present the pathologic changes responsible for this syndrome remain a mystery.

Dr. Kopetzky, in 1933, first suggested to me that attacks of vertigo were most frequent in patients with cardiovasculorenal disease and that this pointed to some effect of disturbed water metabolism which gave rise to a water-logged condition of the static labyrinth. At his prompting, a study of water balance in this class of patients was begun. Other workers had observed that these patients were improved when they eliminated large amounts of water, as by sweating or increased urination. One must, however, consider that body water is not water alone but a solution of electrolytes, chiefly sodium salts, and water. Whenever large amounts of water are lost, the sodium content of the body is greatly decreased. Another point is that "water as such" or the intake of free fluid constitutes only about 50 per cent of the total water available to the body. A surprising amount is lost by vaporization from the skin and lungs. A certain amount of water is lost in stools. The studies, therefore, took into consideration the total amount of water available in and lost from the body. The effect of an increase in the content of sodium and of a decrease in the content through elimination was also studied.

Experiments made on subjects who were subject to frequent and severe attacks led to the conclusion that the symptoms of Ménière's disease are due to the retention of sodium in the body. If a sufficient supply of water is available the sodium is also retained, but this fact is not of primary importance. Apparently, the local tissues involved in Ménière's disease have either an increased avidity for sodium or an unusual sensitivity to it.

The therapeutic indications are: (1) to permit as small an intake of sodium as is possible and (2) to prevent the accumulation of sodium in the body. The former aim is easily attained by means of a controlled diet, and the latter, by the use of acid-producing salts, such as ammonium chloride. When these two factors have been controlled the intake of water need not be considered.

DISCUSSION

DR. S. J. CROWE, Baltimore: Many cases presented in the literature differ from the clinical picture described by Dr. Dandy. His picture is clearcut. His patients all had (1) deafness, (2) tinnitus and (3) attacks of vertigo. The deafness was unilateral in 36 of 40 patients. The 4 patients with bilateral impairment had lesions both of the inner and of the middle ear. In 38 of the 40 patients the deafness was gradual. The loss of hearing was not total but involved both high and low tones. In every patient the bone conduction time for the 512 fork was markedly shortened on the impaired side. Tinnitus was present in the affected ear in 39 of 40 patients. The average frequency of the attacks of vertigo was two a week. Patients with Ménière's disease are completely unnerved and disabled by the attacks.

In comparing these 40 patients with 61 who had verified cases of tumors of the cerebellopontine angle, I found that (1) hearing was totally lost on the affected side in four fifths of the patients with tumors, but not in those with Ménière's disease; (2) in the 13 cases of tumor in which some hearing remained, the results of tests with tuning forks were similar to those obtained for the patients with Ménière's disease, but only 2 patients had similar audiograms. The loss of hearing in cases of tumor of the cerebropontine angle is due to mechanical pressure. A study of 98 cases of tumor of the brain other than tumors of the cerebropontine angle shows that these tumors rarely give rise to a condition resembling the otologic findings in Ménière's disease.

DR. JOHN GUTTMAN, New York: Dr. Dandy said that after severing the eighth nerve he noticed diplopia. In my case a panophthalmitis developed following a severing of the eighth nerve, but I have not been able to see the connection between the reaction in the third nerve and the operation on the eighth.

DR. DOUGLAS MACFARLAN, Philadelphia: If a partial section of the eighth nerve destroys the vestibular branch, relieving the tinnitus, does the hearing on the affected side improve after the relief of the masking effect? What was the future course of the deafness after partial section?

DR. DON M. CAMPBELL, Detroit: I do not believe that the diagnosis of Ménière's disease presents any difficulty, but there is great uncertainty as to the pathologic process of this peculiar manifestation. I believe that Dr. Dandy's work throws light on this side of the question. Dr. Furstenberg's work is interesting. Some of his cases suggest epilepsy. This is borne out by the fact that his patients were benefited by a change toward the acid side of the hydrogen ion content of the blood. In children epilepsy can be benefited by a ketogenic diet, which reduces the alkalinity of the blood and brings about acidosis. This method, however, has the disadvantage of merely bringing about improvement of a condition and not a permanent cure. Dr. Dandy's surgical treatment puts the patient in a condition in which the syndrome is permanently overcome.

DR. FRANK R. SPENCER, Boulder, Colo.: Spasm of the retinal vessels affects the eye. There may also be spasm of the labyrinthine vessels which are temporary and which often disappear in a few hours. I had a patient, a man aged 67, who suffered from dizziness, tinnitus and deafness lasting several hours. Both ears were affected. The blood pressure was 200 systolic and 90 diastolic. He had several attacks. His physician ascribed the condition to spasm of the basilar artery, affecting the labyrinthine branches. Later the patient recovered completely.

DR. EDMUND P. FOWLER, JR., New York: Many cases of Ménière's disease clear up completely if the patients stop smoking and taking alcohol. Apparently there is often an element of toxic neuritis in these cases.

DR. E. M. JOSEPHSON, New York: Dr. Furstenberg attributes the results obtained to limited intake of sodium, and believes that the symptoms are caused by an excess of sodium. He thus markedly affected the amount of calcium. Did he determine the calcium content and the p_H of the blood? Did he consider the p_H of the blood as being too high?

DR. S. J. KOPETZKY, New York: Patients with Ménière's disease come to a general hospital, many of them with symptoms arising from the cardiovascular or renal system, and they often exhibit a gross imbalance of water metabolism. The great majority of such patients would not submit to a surgical procedure such as Dr. Dandy has described. Often, too, the condition is bilateral, and it would be difficult to decide on which ear to operate. In addition, the patients find relief in medical procedures which control the intake and output of water, and which, at present, are often empirically employed. I, therefore, welcome the additional factor which Dr. Furstenberg has introduced into the treatment of such patients. I believe that the cases observed by Dr. Dandy are instances of a distinct clinical entity. The patients treated by Dr. Furstenberg recovered from their attacks with a functioning end-organ, while when the nerve is severed the end-organ is dead. Thus it appears advisable to try medical therapy first, and to resort to surgical procedures only when other therapeutic agents fail.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: While my experience with the medical treatment of Ménière's disease is by no means conclusive, I have been able to produce the attacks by the ingestion of sodium and to relieve the patient by the administration of ammonium chloride. Dr. Campbell has suggested a diet high in protein and it is hoped that acid-producing foods may be of further service in eliminating sodium from the body.

In answer to Dr. Josephson, I wish to state that I have not yet studied the calcium metabolism in these patients. It was demonstrated that sodium is rapidly

excreted in the urine at the time the patient is relieved of his attack and that sodium does not appear in the urine at the time of the maximum seizure. I believe that sodium has an important influence in the production of Ménière's symptom complex.

Evening Session, April 6

DEMONSTRATION OF CATS CONDITIONED TO SOUND. PROF. J. TAIT, MR. S. DWORKIN, MR. G. F. SUTHERLAND and MR. D. A. ROSS, Montreal, Canada.

A simple alimentary-motor conditioning method for testing the hearing of dogs and cats is described. The upper limit of hearing in the dog is 35,000, in the cat, 50,000 cycles, per second. Both animals can discriminate between pure musical notes with respect to pitch and intensity. After bilateral extirpation of the focal auditory area hearing is at first absent; later, the upper range is reduced. Destruction of both cochleae, producing total deafness, does not affect the responses to slight vibrations or body tremors. In one cat bilateral removal of the malleus reduced the upper limit of hearing from 35,000 to about 10,000 cycles per second.

DEMONSTRATION OF CATS CONDITIONED TO SOUND. PROF. E. A. CULLER, Urbana, Ill.

"Conditioning" is a new name for an old and familiar process. It is a specialized form of training. Two things are necessary: (1) a response which is in the animal's repertory and (2) a stimulus, such as hunger, to incite the animal to work. Given these two requirements, many methods are available. In the work at the University of Illinois the incentive is mild punishment (electric shock on the forepaw), and the animal's response is the natural one of withdrawing the foot. The stimulus tone is sounded for two seconds, directly before the giving of a charge strong enough to cause a hasty withdrawal of the foot. The animal soon learns to retract his paw as soon as the tone begins, thereby avoiding the shock. When the tone is fainter he continues to react as long as he hears it; when it becomes inaudible he no longer responds.

When testing human beings one can judge only from the subject's statements what he really hears. The dog reacts by withdrawing his paw. The animal has the incentive to act if he hears the sound. Over a period of weeks the performance of the dog does not vary more than from 1 to 4 decibels. That shows clearly that the animal is not acting at random but is responding only to the tone. These actual tests of trained animals constitute, in my opinion, the court of last appeal in disputed issues.

DISABILITIES CONSEQUENT ON ABLATION OF PARTICULAR RECEPTORS IN THE LABYRINTH OF THE FROG (Motion Picture Demonstration). DR. WILLIAM J. MCNALLY and DR. JOHN TAIT, Montreal, Canada.

The utricular maculae, which are gravity receptors, are stimulated only during movement of the head. Deprived of the vertical canal check, the utricular otoliths are in precarious equilibrium, and their oscillatory movements cause bodily pendulation. The maculae are susceptible to two different kinds of stimulation, slow and quick tilt, only the response to slow tilt being compensatory. When they are quickly tilted in a given direction the uncomplicated maculae give a reflex response identical with that elicited from vertical canals quickly tilted in the opposite direction. During any sudden tipping displacement of the normal animal the vertical canals of one side work in functional association with the utricular macula of the opposite side. The maculae are irresponsive to turntable rotation. Presumably the utricular otoconia acts, not as a weight, but as a gravity-oriented buoy.

From an analysis of turntable rotation it is found that the utricles alone are not stimulated by rotation, yet in their absence the reaction is abnormal. Removal of the vertical canals is followed by unsteadiness of the head during movement.

When either the anterior or, especially, the posterior vertical canal alone is intact there is a definite response to rotation even when the head is held. When only the horizontal canals are intact there is circling, but the head is wobbly; thus the horizontal canals are not sufficient to bring about a normal reaction. The vertical canals and utricles mutally opposed are necessary to maintain the steadiness of the head and to complete the normal reaction.

Morning Session, April 7

SYMPOSIUM ON LABYRINTHITIS SECONDARY TO INFECTIONS OF THE MIDDLE EAR AND MASTOID

PATHWAYS OF INFECTION AND PATHOLOGIC PROCESS. DR. L. W. DEAN and DR. DOROTHY WOLFF, St. Louis.

The clinical investigations in this study were made by Dr. L. W. Dean; the microscopic work was done by Dr. Wolff. The latter work was based on 46 cases of otitis media and on examination of serial sections of 68 ears. Bilateral observations were made in 21 cases in this group. In these both ears were involved, though the involvement was more severe on one side than on the other. Suppurative labyrinthitis was observed in 11 cases. The sections show that the infection may pass from the middle ear to the labyrinth by direct and by indirect extension.

The routes of direct extension are: (1) fistula through the round window, (2) fistula through the oval window and (3) fistula through the bony capsule as a result of (*a*) necrosis of the bone or (*b*) mechanical trauma.

Indirect extension takes place via the blood or the lymphatic vessels. One route does not exclude the other, and the infection may proceed by both means of communication.

Direct Extension.—Fistula Through the Round Window: The round window is deeply placed in a bony niche; in infection a mass of pus collects at that site and drains with difficulty. Nearby is the sinus tympani, another deep, pus-collecting pocket. In infants the petrosal fossula is another adjacent pus-holding structure. The membrane of the round window is also concave and can act as a pus pocket. If the pressure within is increased the membrane can bulge outward into the middle ear. Minute vessels pass from the middle ear to the labyrinthine side. The mucosal layer of this membrane is subject to infection. Sixty-four of 68 ears examined showed pus in the niche of the round window, and infiltration of the mucosal layer of the membrane was present in 43 cases. Nineteen of the 21 bilateral cases showed pus in the niches of the round window in both ears. The fibrinous layer of the membrane of the round window is much more resistant to infection than the mucosal layer.

Fistula Through the Oval Window: The niche of the oval window is in a much more exposed position than that of the round window and can be more readily drained. The stapes articulates with the oval window by means of the fibrous annular ligament, the fibers of which when torn or broken by disease present a surface which allows a port of entry of infection to the labyrinth. In 60 patients examined, pus was found in 51 ears. A fistula through the stapedio-vestibular joint was found in 4 ears. Fistula of the oval window is found at operation more often than that of the round window.

Fistula Through the Bony Capsule Due to Necrosis: This type of fistula occurs most frequently in the horizontal canal and in the promontory. The condition may occur in syphilis or in tuberculosis, with or without cholesteatoma. Without these diseases, chronic necrotic osteitis with cholesteatoma invades the capsule very slowly. Nature tends to wall off the necrotic process, and meningitis rarely results. In an infant acute diffuse labyrinthitis is the result of acute otitis media. When an infected ear and vomiting occur in an infant labyrinthitis should be suspected, as an infant cannot complain of vertigo or deafness.

Fistula Through the Bony Capsule by Mechanical Trauma: This may occur in operative procedures.

Indirect Extension.—Blood Vascular Route: The extensive bed of marrow and the haversian system, especially in infants, constitute a route of direct extension of infection. Some haversian canals surround the cochlea and some penetrate to the endosteum of the labyrinth. The best treatment of labyrinthitis is prevention by adequate drainage. This, however, must be done at a time when nature has built a barrier to the infection. Slow necrotic processes which are already walled off can be dealt with with safety. The best surgical judgment is required to determine the stage at which operative procedure may safely be attempted in cases of otitis of the middle ear and of osteitis for the purpose of preventing labyrinthitis.

Differential Diagnosis. Dr. S. J. Kopetzky, New York.

Labyrinthitis has been classified as (1) circumscribed, (2) fibrinous, (3) serous and (4) purulent. Only the last type may be fatal. Only the types of the disease which occur with tympanomastoid infections are under consideration today. The following points should be established in a clinical diagnosis: (1) a labyrinthine lesion, (2) its differentiation from other intracranial lesions and (3) a differentiation between the serous and the acute purulent type. As perilabyrinthitis may also constitute a threat to life, one must distinguish between the following lesions: (1) perilabyrinthitis, (2) serofibrinous labyrinthitis, (3) hematogenous labyrinthitis, (4) labyrinthine fistula, (5) cerebellar abscess and (6) localized meningitis. One should keep constantly in view as an objective the type of therapy necessary to cure the individual lesion. An important differentiation is to be made between the types of lesions which require an operation on the labyrinth and those which can be healed without surgical intervention.

Perilabyrinthitis.—This condition shows rather transient symptoms, negative reactions of the spinal fluid and normal roentgen findings, except, perhaps a slight cloudiness of the petrosal pyramid. The patient complains of uneasiness, slight dizziness and nausea; often there is deep retro-orbital pain. The first branch of the fifth nerve is affected.

Serofibrinous Labyrinthitis.—In this condition there is dialysis of bacterial toxins which pass through the intact round window. The perilymph is increased, with a consequent increased pressure on the endolymphatic spaces. Hence, violent labyrinthine symptoms ensue—vertigo, vomiting, violent nystagmus directed away from the involved side. The patient is bedridden and lies in the labyrinthine "forced position." The temperature varies according to the amount of otitic suppuration. The severe onset subsides within five days, and the patient is helped by the removal of irritating material from the middle ear. In this form the pressure on Corti's organ produces a loss of hearing acuity. Sound is generally lateralized toward the opposite ear. The spinal fluid sometimes shows a slight increase. Otherwise it is normal, and this fact helps in the diagnosis.

Hematogenous Serofibrinous Labyrinthitis.—This accompanies generalized toxemias, such as scarlet fever and mumps. The cochlear portion of the labyrinth is most affected; usually the canals remain uninvolved. Hence, there are few symptoms of disturbance of the canals. There is a sudden acute loss of hearing; sound is lateralized to the unaffected ear. Reactions to the labyrinthine tests are normal. The spinal fluid is normal, except for an increase in lactic acid. The lesion is the result of serofibrinous exudate and labyrinthine hydrops secondary to the systemic infection.

Fistula of the Labyrinthine Capsule.—This condition is secondary to a lesion of the middle ear which causes resorption of bone. It occurs most frequently with cholesteatoma, in which the horizontal semicircular canal is most often involved. It may also result from acute necrotic otitis. The patient suffers attacks of dizziness which increase in severity. The labyrinthine tests give exaggerated

responses. The diagnostic data include a history of long-standing, persistent otorrhea with a lesion of the middle ear of a dangerous type, whether the fistula test is positive or not. There is dizziness but normal cerebellar equilibratory cerebellar function.

Acute Generalized Purulent Labyrinthitis.—All the preceding types may develop into this type. The patient presents a violent nystagmus directed toward the affected side, with frequent spells of vomiting and vertigo. He lies in the "forced position" and has high fever. Sound lateralizes toward the uninvolved ear. The labyrinth fails to respond to stimulation. The hearing acuity is lost. In the course of time the severity of the symptoms abate. The patient is able to relax, but the fever and headaches remain marked. Then the condition either resolves, with recovery but permanent damage to the ear, or progresses rapidly toward meningitis. At first the meningeal reaction may be considered protective, and the observations on the spinal fluid are very important. The lactic acid is increased, and the ratio between the lactic acid content of the fluid and that of the blood plasma is also increased. The chloride, carbonates and p_H of the fluid drop below their normal gradients in relation to the blood plasma. The pressure on the arachnoid is evidenced by the presence of cells, globulin and albumin.

At this point, one must differentiate the condition from cerebellar abscess, which also gives postoccipital headache, nystagmus, vertigo and vomiting. It may also show the "forced position," but this is usually absent. In cases of cerebellar abscess the reaction to the Weber test is lateralized to the diseased ear; the hearing is not lost; the caloric tests are normal, and the nystagmus starts gradually and persists until death or termination. The cerebrospinal fluid is under increased pressure. When distal signs of ataxia and disorientation are present the presence of a cerebellar abscess is more certain.

Finally, a collection of pus in the pontile angle system may simulate symptoms of purulent labyrinthitis. Nystagmus is variable; its direction varies; vertigo varies, and vomiting may be present. The hearing is not lost; sound is lateralized to the involved ear. The labyrinthine tests show a reaction to stimulation. The patient usually prefers to lie on his back. The proportion of lactic acid in the spinal fluid is increased. The gradient between the blood plasma and the cerebrospinal fluid for carbonates, chlorides and p_H is lowered. The globulin and albumin contents are increased, and in later stages bacteria are present in the fluid. If the infection persists a typical meningitis develops, at which time bacteria are usually found in the spinal fluid.

TREATMENT OF LABYRINTHITIS. DR. I. FRIESNER and DR. H. ROSENWASSER, New York.

This article was published in full in the August 1934 issue of the ARCHIVES, page 139.

DISCUSSION

DR. ARTHUR B. DUEL, New York: If the subject of labyrinthitis is cleared of its limbs and other flourishes and viewed from the surgical standpoint it does not present so many difficulties. No one, including the men who have read papers today, has had sufficient experience to establish autocratic conclusions, but if the minds of otolaryngologists were clearer as to what might be done the patients would be benefited more than if the physicians vacillate between one method and another. No matter how large a man's experience may be, a case of labyrinthitis is always an exciting adventure, a cataclysm which calls for quick decision, for the results rest on that decision. A case of labyrinthitis is either operative or nonoperative. Acute labyrinthitis is never operative at a certain stage. No matter what the case, I should not operate before the process extended beyond the confines of the labyrinth and threatened to produce meningitis.

There are two types of acute labyrinthitis regarding which there is a wide divergence of opinion. Acute traumatic labyrinthitis, after puncture of the ear drum or after operation on the mastoid is apt to present violent symptoms. No

one, however skilful, has any certainty that he is going to avert the catastrophe of meningitis. Any one who adds to the menace by additional traumatic manipulation may bring on the very catastrophe he is trying to avert. I give sedatives, avoid annoying the patient by too many tests and allow him to pass on into the latent stage for from forty-eight to seventy-two hours. I am guided by subsequent manifestations as to whether the patient should be operated on early or left alone until a later stage, which may be regarded as chronic labyrinthitis and in which the condition may be studied and as many examinations made as desired.

One important observation in labyrinthitis is whether there is any hearing on the diseased side. If any hearing is left operation should not be performed unless one fears meningitis. Few otolaryngologists have seen cases of meningitis in which, judging by the photophobia, Kernig's sign and ataxia, there is any question that operation is necessary and that the condition is not labyrinthitis but meningitis in which the labyrinth will have to be exenterated. I seriously disagree with those who perform a spinal puncture to determine whether or not meningitis is present. If the patient shows signs of intracranial complications, I order him to the table and just previous to operation make a spinal tap. I think that there is a possibility of lowering the intracranial pressure and adding to a slight possibility of admitting extension of the suppuration into the subarachnoid space.

At the present time one cannot say whether or not the intracranial pressure is not of the greatest aid in preventing extension of the meningitis. In the acute stage the patient should be disturbed as little as possible. In operating, it is of advantage to use the hammer, chisel and gouge as little as possible. The operation, when done, should be the simplest one that will allow drainage of the labyrinth.

Dr. J. M. Smith, New York: In former articles on labyrinthine surgery I have called attention to the fact that too many labyrinthine operations are being performed, that the mortality is too high and that nature, if given a chance, will wall off infections between the labyrinth and the meninges by barriers of plastic exudate or granulations. This means that a large percentage of patients will recover by means of a complete radical operation on the mastoid and a conservative Hinsberg drainage of the labyrinth. Clinical experience since that writing in 1922 has proved that these principles are sound and that it is not the labyrinthitis but the resulting meningitis that kills the patient. The problem, then, is to check the labyrinthitis before meningitis occurs.

Anatomically the cochlea and vestibule form a good part of the internal wall of the middle ear; the round and oval windows and the horizontal semicircular canal are constantly bathed in infectious secretion, and while invasion of the labyrinth may occur through any portion of the wall, it usually gains access through one of these areas. Fortunately most infections of the labyrinth are due to chronic, necrotic processes, which give nature a chance to form a protective barrier of granulations.

Sudden acute invasion of the labyrinth by the blood stream is more dangerous because there is no time to seal off the communication of the perilymph with the spinal fluid or the endolymph from the arachnoid space.

Labyrinthitis may be divided into the following classes: (1) perilabyrinthine infection, (2) circumscribed labyrinthitis, (3) diffuse latent labyrinthitis and (4) diffuse manifest labyrinthitis. In the first type symptoms are due to irritation from extracapsular infection with no impairment of function. A simple or radical operation is all that is indicated.

Circumscribed labyrinthitis is most often due to erosion of one of the canals, usually the horizontal. A test generally shows a partially functioning labyrinth. A radical operation should be performed and the case followed closely. Secondary drainage of the labyrinth by the Hinsberg method should be performed if the case demands it.

In the diffuse latent type the labyrinth is dead, with no active symptoms. I believe that if the labyrinth is dead and the ear dry nothing should be done. If there is a discharge, with foul odor, an examination of the spinal fluid should be made. If the fluid is normal, a radical operation should be performed. If no

necrosis is found nothing more should be done. If necrosis is present a Hinsberg drainage should be done. Care is taken not to destroy the barrier by opening through the second turn of the cochlea and establishing a communication through the spinal fluid.

In the purulent diffuse type there is a dead labyrinth with symptoms of acute labyrinthitis. The first step is spinal puncture, and if the fluid is normal, one should wait and watch and not attempt an operation. Many such cases subside without intracranial operation. If there is an increase in the cell count of the fluid with no bacteria a Hinsberg operation is sufficient. With bacteria in the fluid a complete operation is indicated, including the making of an opening into the internal auditory meatus with incision into the dura in the posterior fossa; with such a condition there is little hope of recovery.

Traumatic labyrinthitis is usually due to injury. I believe that rest in bed under observation is best. If diffuse meningitis follows, an operation is likely to be of little use, while patients sometimes recover from the stormy symptoms following the injury.

Dr. H. I. Lillie, Rochester, Minn.: This symposium has shown that the differential diagnosis between acute diffuse suppurative labyrinthitis and acute diffuse serous labyrinthitis in the very early, acute stage may be difficult or impossible. I think that the symptoms of the suppurative type subside more quickly than those of the serous type, probably because the suppurative process destroys the end-organ.

It seems fairly well established that operation may be deferred during the active, acute stage unless signs of meningeal involvement occur. If there are no signs of meningeal involvement, the Kerrison and the Hinsberg operations and other operations of this type have given good results. If, however, an extensive pathologic process is found and meningeal involvement occurs, the more radical Neumann operation is called for. Not all the patients, however, who present symptoms of labyrinthitis, need be subjected to an operation on the labyrinth.

Of the last 100 cases in which I performed the radical type of mastoid operation, definite lesions of the labyrinth were found in 23. Erosion of the canal without fistula was found in 5 cases; fistula, single or multiple, was found in 18. Three patients with fistula had serous meningitis; 1 had a cerebellar abscess. The Neumann operation was used in 8 cases and the Hinsberg in 6. All the patients recovered.

Dr. L. W. Dean, St. Louis: Dr. Smith spoke of a fistula through the canal, but in following the fistula after an operation on the mastoid, if there is a leakage the fluid collects in the tympanic cavity. How long does he allow the fistula to drain?

Dr. S. J. Crowe, Baltimore: It is unfortunate that these papers and discussions are limited to labyrinthitis in adults, because the condition occurs frequently in children and is often very difficult to recognize. It is humiliating to otolaryngologists to see in the autopsy room a purulent labyrinthitis which has not been suspected.

Dr. L. W. Dean, St. Louis: There is such a thing as serofibrinous labyrinthitis of the endolymphatic space. Occasionally one sees in the laboratory a diffuse purulent labyrinthitis involving the perilymph with typical serofibrinous labyrinthitis of the endolymph. It has never been demonstrated, however, that serofibrinous labyrinthitis is not a mild infection rather than a toxic condition. New bone may form very rapidly in the labyrinth. New bone may develop from the endosteum within eight weeks following the initial infection.

Dr. Samuel J. Kopetzky, New York: I do not wish to be understood as representing lumbar puncture as a totally harmless procedure. I suggest that when it is used the reaction in the meninges be ascertained, that it be performed under all safeguards and that only enough fluid be taken to make the required tests. It should be properly performed and properly safeguarded.

I should not base surgical procedures on laboratory examinations alone. The latter, coupled with the bedside clinical picture, give differentiations of extreme value in determining the status in a given case. I believe in waiting for the "walling off" process to take place. If the tests of the fluid give evidence that the infection is reaching the meninges, some action is necessary. In operating on such patients I try to produce the minimal surgical trauma and do not use a chisel, hammer or gouge. I believe that labyrinthitis in children differs little from that in adults, except that children are unable to complain of vertigo and functional tests are extremely difficult. In reference to the discovery of such cases at autopsy, it may be that the labyrinth is often invaded from its visceral side, from the meninges, rather than from the middle ear. These deaths are directly from meningitis, with no significant lesion in the mastoid. I agree with the speaker who said that he sees too many labyrinthectomies. There is a time for watchful waiting, and if the fluid gives reassuring signs no surgical intervention is needed.

DR. I. FRIESNER, New York: My conclusions are not in any sense authoritative, but I have formulated a certain plan of procedure, and I am glad to find that, in the main, the speakers agree with me. I believe that the membranous barriers of the labyrinth can admit toxins and yet exclude organisms. I use lumbar puncture with caution in cases in which the symptoms indicate that there is an extension of the infection beyond the confines of the labyrinth, but I have never been a proponent of repeated lumbar puncture.

Afternoon Session, April 7

SYMPOSIUM ON MENINGITIS SECONDARY TO OTITIS OR SINUS INFECTION

A COMPREHENSIVE STUDY OF OTITIC MENINGITIS. DR. JOSEPHINE B. NEAL, DR. EMANUEL APPLEBAUM and DR. HENRY W. JACKSON, New York.

Our studies comprise 623 cases of meningitis, excluding the meningococcic and tuberculous types and stressing particularly the clinical aspects. Our tables show the relation of cases to primary sources of infection. In table 1, which gives a complete list of cases, infections of the ear and sinus are not conspicuous. In 9 cases there was a history of a probable primary focus of infection. Table 2 lists conditions regarded as primary in the more common forms of purulent meningitis. Our studies show that meningitis secondary to mastoiditis or otitis media is most often due to streptococci, but that the form secondary to respiratory infection is most frequently due to pneumococci and to influenza bacilli. In our series injuries to the head and skull have been an infrequent cause of meningitis. Table 3 shows typing of pneumococci (by agglutinin and precipitin tests) in relation to etiology. Type III was most common in meningitis secondary to otitis media, mastoiditis and sinusitis, while type I was more commonly associated with respiratory infections. Table 4 gives a grouping of streptococci (by red blood agar) and shows that meningitis is most commonly caused by the hemolytic streptococci. Table 5 gives a grouping of the staphylococci found, and it is noted that Staphylococcus aureus predominates. It was noted that meningitis, apparently secondary to a primary focus, did not always show the same organism cultured from the focus.

The symptomatology combines the general reaction to acute infection and the specific reaction of the involvement of the meninges and central nervous system. The onset is usually sudden, but it is often masked by symptoms related to the primary focus. Early observations on the spinal fluid are often inconclusive. The symptoms may be mild or exceedingly violent. With the progress of the disease convulsions occur, especially in children under 2 years. In infants the signs of meningeal irritation are slight and hard to ascertain definitely. In older patients stupor is more frequent than delirium. Both the knee jerks and the reaction to light tend to become lost as the case progresses. In influenzal, streptococcic and staphylococcic cases a hemorrhagic rash is often seen. In influenzal

forms the temperature reaches a peak (107 F.) before death. The course of purulent meningitis is often very rapid, sometimes lasting only forty-eight hours, but it may last for four or five days.

In several cases we have seen marked remissions with ultimate recovery. Sixteen patients have recovered: Nine had an infection with streptococci; 4, with influenza bacilli; 1, with staphylococci; 1, with Friedländer's bacillus, and 1, with both meningococci and staphylococci.

On examination the fluid is usually turbid and under increased pressure. In fulminating cases the fluid is sometimes clear but shows an overabundance of organisms. The globulin and albumin are increased. The sugar decreases and later may disappear. The bacteriologic study of the fluid is highly important. The organism should be obtained by culture.

Blood cultures were made in 68 cases; 32 were positive. Thirteen of 38 aural cases showed positive cultures. One cannot always tell whether bacteriemia follows or precedes the meningitis. It adds to the gravity of the prognosis.

Serous meningitis is often found with otitis media. Well marked signs of meningeal irritation are present. The spinal fluid is cloudy, with increased cells, albumin and globulin and with normal sugar. No organisms are found by smear or culture. This condition may progress to true bacterial meningitis.

The mortality in meningitis is so high that we believe that education of the public as to the danger of the neglected common cold and as to the early treatment of infections of the nose, throat, ear and sinuses is imperative, as is also the prompt and thorough eradication of primary foci of infection. In addition to this an adequate drainage of the cerebrospinal fluid is recommended as a therapeutic measure. In 23 cases forced drainage has been recommended and used, but in some instances the autopsy showed extensive edema of the base of the brain. Great care, therefore, must be exercised with this procedure. Our results with specific serums have been discouraging. We continue to use them in the hope that more potent serums may be developed in the future. Autogenous vaccines have also been used. In 2 cases recovery followed, but the course of meningitis is usually so short that this measure cannot be used. In our opinion chemical agents, such as gentian violet, a mixture of equal parts of acriflavine hydrochloride and gentian violet, acriflavine hydrochloride and optochin hydrochloride are of doubtful value and are often harmful or even dangerous.

In conclusion, the difficulty in treating meningitis is due to the numerous foci of infection, which cannot all be eradicated and cause constant reinfection of the meninges. The variations in the virulence of bacteria and in the reactions of the host are unpredictable factors. Often recovery may follow infections by bacteria of low virulence and those in which the host is able to cope with the organism. No specific procedure can, therefore, be formulated. Early and repeated spinal drainage may be regarded as important; specific serums may be employed intraspinally; surgical measures may be employed to remove foci, but further than this, therapeutic measures must be regarded as experimental. One must bear in mind the possibility of spontaneous recovery.

BACTERIOLOGY. DR. ANNA W. WILLIAMS, New York.

Of the 597 cases of meningitis, other than those due to meningococci or tubercle bacilli, 205 were due to streptococci, 214 to pneumococci and 118 to influenza bacilli. The great majority of these cases occurred after infections of the middle ear and sinuses.

The importance of determining the serologic types of these microbes is emphasized. In the case of the pneumococcus the identification of type is on a satisfactory basis. With the influenza bacillus some advance has been made, but little is known about the incidence, either seasonal or geographic, of agglutinative types among hemolytic streptococci, particularly in these cases. And until such knowledge is obtained one cannot decide how much worth while it is to prepare antibacterial serums of a particular type.

Thirty-seven strains of streptococci from cases of meningitis were studied: Twelve of them fell into the scarlet fever serologic groups and two into their erysipelas type 1 group, and seven formed a new agglutinative group.

Therefore, in order to test the worth of preventive and curative measures in treating patients with otitis media and related infections with invasive forms of micro-organisms, one must know much more about the agglutinative types of the organisms producing such infections.

PATHWAYS OF INFECTION. DR. ANDREW A. EGGSTON, New York.

The point of view presented is that of a pathologist associated for fifteen years with a hospital for diseases of the eye, ear, nose and throat. This position has given him a unique opportunity for the study and observation of suppurative meningitis.

A review of the cases of intracranial suppurative diseases for the past eight years at the Manhattan Eye, Ear and Throat Hospital has been made. This includes an analysis of the deaths which have occurred during the period from 1926 to 1933 inclusive. In that time there were 363 deaths; of these, 110 were from suppurative meningitis; 53 from abscess of the brain, and 14 from both abscess of the brain and meningitis. Thus, there were 177 cases of suppurative intracranial disease—48 per cent of all the deaths in the institution. One hundred and seven occurred in males, and 70 in females. The majority of cases occurred between 1 and 10 years of age.

Recovery occurred in 8 cases with clinical symptoms of meningitis, although there was doubt as to the active suppurative process. There were 10 patients with abscess of the brain who recovered.

The following tabulation shows the number of suppurative cases with the pathways of infection:

Total deaths for eight year period between 1926 and 1934			363
Deaths from intracranial suppurative disease			
1. Meningitis	110		
2. Meningitis and abscess of the brain	53		
3. Abscess of the brain	14		
	177	48.8%	
Pathways of suppurative meningitis			
1. Auditory apparatus	90		
2. Paranasal sinuses	18		
3. Cavernous sinuses	5		
4. Postoperative	8		
5. Miscellaneous	3		
	124	70%	
Pathways of abscess of the brain			
1. Auditory apparatus	37		
2. Paranasal sinuses	18		
3. Osteomyelitis, etc.	3		
4. Miscellaneous	9		
	67	30%	

Distribution as to Age and Sex

Age	Cases	Male	Female
1-10	42	107	70
10-20	33		
20-30	31		
30-40	20		
40-50	28		
50-60	18		
60-70	5		

Many pathways may convey infection from adjacent soft or bony structures to the meninges, and infection may travel slowly or rapidly. Often intracranial infection occurs with more superficial pathologic changes and is acute and fulminating; so death occurs rapidly. In other cases the infection spreads more slowly, but the prognosis is always bad.

The infection may be localized as an epidural or subdural abscess, but if pus is not drained the smaller vessels convey it into the brain, and abscess of the brain or meningitis results. The prognosis depends on the pathways and on the rate of the invading bacteria. In frank meningitis, with a spinal fluid cell count above 3,000 per cubic millimeter, with bacteria by culture and with a loss of sugar, the prognosis is very poor.

The cases in which the spinal fluid count is below 3,000, with bacteria present by smear but not by culture and with only a slight change in the amount of sugar, represent a soiling of the membrane by closely adjacent inflamed structures. However, if the primary process does not subside, the bacteria reach the dura and arachnoid, and true meningitis results.

Close cooperation between the pathologist and the surgeon is necessary in order to understand the processes of intracranial suppuration. The pathologic processes in the skull are analogous to those in the abdominal cavity. For years surgeons have known how to treat suppuration of the peritoneum. The primary focus is removed in order to prevent the spread of the disease to adjacent organs. Intracranial suppuration should also receive radical surgical treatment; otherwise, injections or drainage are of little avail.

PRESENT STATUS OF TREATMENT. JAMES G. DWYER, New York.

I noted the remark of Dr. Williams as to Streptococcus mucosus-capsulatus that "there ain't no such animal." For fifteen years I have been preaching and teaching that statement. I should like to endorse the exact bacteriologic work done by Dr. Williams on the types of pneumococci. My experience with meningitis coincides with that of the other speakers. Occasionally, recovery occurs, and one wonders what brought it about. In twenty-seven years I have seen 384 cases of meningitis, which I have tried to follow. There were 16 recoveries; 2 occurring without any form of treatment. In the other cases three agents were used: (1) methenamine, (2) antistreptococcus serum and (3) strained leukocytic extract. Dr. Zabriskie will remember another case. It was thought to be due to an infected lumbar puncture needle that had not been sterilized. The case was very severe. The spinal fluid was red with streptococci. In that case antistreptococcus serum was used. The patient recovered after a stormy course. Another patient, a girl, had a fulminating type, with delirium and a temperature of 106 F. She and some other patients got well. I do not know why.

I take it that this symposium deals with diffuse meningitis. One gets bewildered on reading the various reports in the literature. One type is described with cloudy spinal fluid but no organisms. I do not think that this is the type under discussion. There may be two types of meningitis in which organisms are present in the spinal fluid. In one the organisms are growing like a true culture; in the other they have seeped down from the meninges but are not growing. Recovery occurs in the latter cases, and the organisms are intracellular. Organisms found in the smear that do not grow are damaged and are undergoing autolysis.

There is an analogy in nature which shows why one should differentiate with organisms and not with clear or cloudy fluid in meningitis. In corneal ulcer, one may find pneumococci or streptococci. The anterior chamber of the eye is filled with pus, but it is sterile pus, or rather an exudate of leukocytes. The keratome is used and the pus evacuated. This is a reaction of the tissue to organisms on the outside. Dr. Friesner raised the question of how organisms remain outside a membrane, but the toxins penetrate. I have found organisms on the outside of the dura. This is nature's bulwark. In meningitis a leukocyte count as high as between 30,000 and 40,000 is found.

The treatment of meningitis may be (1) surgical, (2) chemical, (3) serologic, (4) physical and (5) combined.

The surgical treatment may be of one of two types: (1) removal of the original focus and (2) treatment of the meningitis by surgical measures. The first of these two methods is justifiable, but the second is not. One does not have to operate in every case of meningitis. When nature is sealing the infection, the original

focus can be removed. In the rapid forms there is no original focus. The more surgical intervention is used, the more harm is done. Some of these cases may be due to sphenoid involvement. In such cases operation is through the roof of the sphenoid sinus. Many cases are due to osteomyelitis. This travels through the floor of the middle ear. Mastoidectomy does not help in such cases; they usually progress to meningitis.

Surgical intervention, especially the establishment of drainage, has a place. I believe that opening the dura opens up pathways for infection. One can drain the sylvian fissure or the ventricles. There is not enough evidence as yet about drainage of the cisterna magna.

Chemical treatment may include the use of methenamine, optochin hydrochloride, insulin or iodine.

For physical treatment, the Kubie method may be used.

Serologic work on pneumococci and streptococci has been done, but these organisms are very heterogeneous. The treatment is doubtful. Lastly, there is the combined treatment. In conclusion, one must beware of being overpessimistic. Meningitis is the most tragic condition in otology. The mortality is very high. I believe that the most promising field for the future is the development of an antiserum for the individual organism.

TREATMENT BY FORCED DRAINAGE. DR. LAWRENCE C. KUBIE, New York.

The theoretical basis for the method of forced drainage lies in the following fact: When the intracranial pressure is lowered to atmospheric pressure and when at the same time the osmotic pressure of the blood is reduced by intravenous injections of hypotonic saline solution, and simultaneously cerebrospinal fluid is allowed to drain from the lumbar subarachnoid space, it is found that the cerebrospinal fluid is formed not from the choroid plexus alone but by transudation through all the capillaries of the central nervous system. This newly formed fluid then travels along the perineuronal and perivascular spaces to the subarachnoid space and out through the lumbar needle. This affords a natural process of drainage which influences the accumulations of inflammatory products and the course of infectious processes without increasing the intracranial pressure or causing hydration of the parenchymatous tissues and without overburdening the circulations in any way. Forced drainage is thus a true perineuronal and perivascular drainage and not merely a process of evacuation of the preformed lake of subarachnoid fluid, such as is accomplished by simple drainage alone. Furthermore, simple drainage is a self-limited procedure because the collapse of the leptomeninges around the central axis of the central nervous system soon obstructs further drainage.

A trial of forced drainage is indicated in the presence of any acute or chronic infection of the central nervous system. It is contraindicated whenever one suspects an intracranial lesion into which fluid can be poured without an opportunity for free escape. It is also to be used only with caution, if at all, in the presence of cardiorenal deficiency or of active inflammation elsewhere in the body. And, finally, it may be contraindicated when active infection exists in the blood stream. Whether or not it is true, as the experimental work of others has suggested, that infection can be carried from the blood stream into the cerebrospinal fluid under those circumstances remains to be proved in terms of actual experience with patients.

When dealing with patients in whom a pyogenic meningitis has resulted from an infection of the ear or of the sinuses, it may at times be advisable to attack the meningitis first by forced drainage, then to clear out the infected bone and at the same time to continue the drainage throughout the operation and for some period thereafter.

In using this procedure the purpose of all technical manipulations must be to reduce the intracranial pressure to atmospheric pressure as rapidly as can be done without undue discomfort to the patient. This rate varies from one person to another. Hypotonic saline solution is administered in large volume, varying from 1 to 3 liters in the course of from one to three hours, and sometimes beginning during the initial reduction of pressure and sometimes not until after. When

headache comes on early the hypotonic solution must be administered early. The concentration is about 0.45 per cent, but it can be less when fragility of the red cells permits the injection of weaker solutions. The duration of the period of drainage varies from one hour to several hours or even several days. The frequency varies from once a day to once a week.

The results of treatment in 10 cases are presented. Two patients with pneumococcic meningitis, with heavy infection of the blood stream, died. A third patient with pneumococcic meningitis, without infection of the blood stream, showed a marked, though transient, improvement, but, also died in the end. A fourth patient with pneumococcic meningitis, without infection of the blood stream, made an excellent recovery. Two patients with meningitis due to a hemolytic streptococcus, but without infection of the blood stream, recovered. Three cases of severe meningeal reaction, without the demonstration of organisms in the cerebrospinal fluid, but following chronic infection of the middle ear or sinuses, terminated in 2 recoveries and 1 death. And, finally, a case of a mistaken diagnosis is described, in which a cerebellar abscess was thought to be oncoming meningitis and was treated as such, as a result of which acute symptoms of pressure developed, and the patient died of respiratory failure.

DISCUSSION

DR. WELLS P. EAGLETON, Newark, N. J.: The treatment of meningitis has been based too much on drainage and the classifications too much on etiology. Many of these preconceptions must be abandoned. Otolaryngologists operate in desperate terminal cases, hoping to find something that may be amenable to surgical intervention. On the other hand, they delay in opening the dura early, when there is a fair prospect of success, because they fear that their localizing diagnosis is incorrect and that the patient may be suffering from an extradural suppuration on which meningitis may be grafted. This is a sad commentary on the knowledge of specialists.

Any patient who has suppuration in the ear with pain in the head at night should be taken to a hospital and carefully observed. The routine examination should include repeated roentgenograms of the apexes, a daily differential leukocytic count and a Schilling count, as well as a perimetric examination of the visual fields.

Suppurative meningitis is not necessarily associated with a high cell count and with micro-organisms in the fluid from the lumbar region. I have seen cases in which micro-organisms in the subarachnoid spaces were associated with a clear, sterile fluid and with a small cell count from the lumbar region. I have often taken infected fluid from the basal cistern when the lumbar fluid was sterile. Therefore, one cannot wait for a high cell count in the spinal fluid to make a diagnosis of meningitis.

In my series of over 400 cases of local or general suppurative meningitis, originating from the ear and nose, 24 originated from the sphenoid, and 42 from an infection of the petrous apex. About 32 per cent of the patients operated on recovered.

There are two distinct varieties of suppurative meningitis, depending on the mode of invasion of the infection and on the method of extension in the arachnoid: (1) meningitis of the subarachnoid space and (2) meningitis secondary to infection of the pial vessels. Meningitis of the subarachnoid space may be cured surgically as long as the infection is limited to the basal cistern. Meningitis due to an infection of the pial vessels is uniformly fatal, at least when it has advanced beyond the primary focus.

The usual form of suppurative meningitis from either the ear or the nose is subarachnoid meningitis. It may be cured surgically if the diseased focus near the dura is removed. The primary focus in the bone may be a diseased petrous apex (often an unrecognized source of meningitis). Infection of the mastoid of the lateral sinus and osteomyelitis of the frontal bone may be cured surgically in a large proportion of cases. A symptomless infection of the sphenoidal bony base is a frequent cause of meningitis, especially of the pneumococcic type. In meningitis

due to Pneumococcus type II, I have obtained a cure by simply draining the sphenoid sinus and administering serum. I consider that the red bone marrow of the sphenoid has a distinct bactericidal effect.

In my experience operation on the meninges gives a high percentage of recoveries if it is preceded by ligation of the common carotid artery, because ligation of the carotid artery relieves venous congestion, which at autopsy is always found in meningitis.

The more severe form of meningitis, "blood vessel meningitis," occurs within the vessels of the meninges and brain; it is associated with bacteriemia. No form of treatment has been successful with this phlebitic type of meningitis. In these cases, injection into the carotid artery (Kolmer) and forced drainage (Kubie) may be tried. The most important curative treatment is early recognition and drainage of the primary focus.

Dr. Edwin G. Zabriskie, New York: Unless the focus of infection is removable all methods must fail. In many cases, the focus empties directly into the subarachnoid space or is in that space. All otolaryngologists have been confronted with this tragic situation. In the case reported at the Neurological Institute an attempt was made to introduce corrosive mercuric chloride directly into the subarachnoid space. The patient got well but after a very stormy and violent course. At one time there was a system of flushing the ventricles through the lumbar sac, but only a small proportion of patients were saved. When one considers the complicated and intricate form of the meningeal system it is easy to understand how few of these patients can survive.

In regard to Dr. Kubie's work, Dr. Hare and I have tried his method in cases of pyogenic infection of the central nervous system, and the results have been dramatic. Our last few cases have been disappointing, it is true, but we were encountering formation of various foci of infection that no method could reach.

Another point brought out was that one is often surprised at the vitality of the patient in the face of intense toxic absorption. Thus anything that stimulates the patient to exercise his own powers of resistance will be of benefit. I hope that with the use of new methods a great deal more will be learned about these cases in the next few years.

Dr. Andrew Eggston, New York: If red bone marrow is capable of destroying bacteria, that is news to me. It does not seem to do so in osteomyelitis of other bones. Infections of the head of the femur often lie dormant for many years and then flare up again. The structure of the petrous bone is complicated, and sometimes pneumatic cells are mistaken for cancellous spaces, but pus in the spaces should be easily differentiated from the normal cellular elements by microscopic examination.

Dr. I. Gordon Wilson, Chicago: What fills the spaces?

Dr. Andrew Eggston, New York: Fat and red bone marrow, with some fully developed red and white cells.

Dr. Alfred Lewy, Chicago: I have seen 5 cases of otitic meningitis in which forced drainage was performed by Levinson. They were all late cases and ended fatally. The ordinary hammock is now used instead of the Bradford frame, as it is more comfortable for the patient. I have seen a case of meningitis, secondary to otitis media and mastoid operation, with hemolytic streptococci in the spinal fluid. The patient is still alive on the forced drainage system. I think that the treatment is an advance so far as the comfort of the patient is concerned. I hope to get cases in a sufficiently early stage to report recovery. Hypertonic solution of dextrose has been tried with some apparent temporary improvement.

Dr. Josephine B. Neal, New York: I do not think that one can diagnose bacterial meningitis unless the organisms are demonstrated by smear and culture. Serous meningitis often gives marked symptoms, but there are no organisms and no decrease in sugar. The decrease in sugar is important in making a diagnosis. Recovery takes place after the eradication of foci.

DR. SAMUEL KOPETZKY, New York: When one studies autopsy material in detail, one finds a number of specific lesions in the meninges, the dura, the pia and the cortex, all of which give a general composite picture, clinically regarded as meningitis. When it is possible to evaluate better by bedside study the lesion a patient presents, one will see further daylight. Obviously, a thrombosis of the pial vessels with sepsis is different from an infection of dural connective tissue. Again, a cerebritis differs from the other types. I believe that otolaryngologists are fortunate to have Dr. Kubie's work for bedside use. I hope that it will give a therapy adequate to deal with meningitis of otitic origin. In all my experience, the organism most generally encountered in these cases is the hemolytic streptococcus. Staphylococcus has been reported today, but I have never encountered this organism.

DR. EDWARD P. FOWLER JR., New York: What effect do the hypotonic solutions have on the cytology of the blood other than the diluting effect?

DR. J. GORDON WILSON, Chicago: The pneumatic cells in the mastoid are air spaces with membranous linings. The cells in the petrous tip, on the contrary, are lined with red bone marrow. Therefore, an infection of the petrous tip ought not to be regarded as a direct extension from the pneumatic cells of the mastoid or the pneumatic cells about the eustachian tube, but as an infection of the blood.

DR. LAWRENCE C. KUBIE, New York: An important point is the significance of bacteria in the cerebrospinal fluid, as stressed by Dr. Neal and Dr. Williams. Unquestionably, when bacteria are absent in the fluid the disease runs a milder course. Nevertheless, I doubt whether the absence of bacteria proves that the condition is not a true meningitis. The cerebrospinal fluid is peculiar among body fluids in several ways. Cells break down in the spinal fluid more rapidly than in other fluids. It is very low in proteins and in all buffer salts. When exposed to the air it becomes alkaline more rapidly. By the local breaking down of sugar it becomes more acid than other fluids in the body. Therefore, the problem of survival of bacteria is a complicated one. The meningitis may be due to virulent organisms which are rapidly destroyed. Perhaps special precautions are necessary in making smears and cultures from the spinal fluid. This is merely a chemical opinion. I am not a bacteriologist.

A hypertonic solution of sugar is of doubtful value. Sugar is either oxidized or stored in the liver. The use of a hypertonic solution is more rational, as one gives large quantities of fluid and can use as much sugar as desired and at the the same time produce the desired dilution of the blood. Hypotonic solution of sodium chloride definitely produces a drop in the electrical conductivity of the blood and a dilution of nondiffusible colloids which forces the water and diffusible electrolytes to leave the blood stream rapidly. The optimal manner of injection —whether to give one long, slow injection, or short repeated rapid injections— is not yet known. This is a matter for clinical experience.

INFLUENCE OF FLUORINE ON THE BONY LABYRINTH OF THE WHITE MOUSE. DR. ALFRED LEWY, Chicago.

This article appears in full in this issue, page 643.

DISCUSSION

DR. EDMUND P. FOWLER JR., New York: Grahe and Greibel (just mentioned by Dr. Lewy) have a technic for diagnosing otosclerosis whether deafness is present or not. They mix the serum of the patient with ground-up normal labyrinthine capsule. The amount of calcium which dissolves in the serum of the otosclerotic patient is greater than that which dissolves in the serum of a normal person. Phosphorus works out similarly. Unfortunately, all their patients with otosclerosis had a low blood calcium, which might account for this phenomenon. However, this is the type of experiment which is necessary to the work on otosclerosis clinically. It is interesting that different agents produce a process

similar to that found in otosclerosis. Venous stasis, infection, overdosage of viosterol, overdosage of parathyroid extract and a diet low in calcium without vitamin D all cause it. My father and I believe that infection plays a part in the causation of otosclerosis.

DR. L. K. GUGGENHEIM, St. Louis: Any experimental work which produces change in the labyrinthine capsule is important. Grahe and Tobeck have found that the capsule is resistant to changes, more so than other parts of the body. Usually the oval and round windows and the cochlea have shown no changes in the experimental work. These changes in metabolism appear to be without significance as causative factors in otosclerosis, and there is more ground for accepting the view of Dr. Fowler that inflammatory processes play the most important rôle.

DR. C. C. BUNCH, St. Louis: A woman, aged 27 has been observed for two years. She was first seen in July 1932 and next in September of that year. The second visit showed an additional loss of hearing. She was hospitalized for complete examination, and at that time her basal metabolic rate was minus 7, and she suffered from being unusually cold and chilly. She was given parathyroid extract while in the hospital. After being discharged she had an abnormally low temperature (95 F.) in the morning, which became normal later in the day. Tests for tuberculosis gave negative results. Her basal metabolism was sometimes minus 22. She was given thyroid extract. Dental films showed two impacted and unerupted third molars. She is up and around, but her weight is below normal.

A woman, aged 56, was observed for two years. She was diabetic and came in for insulin therapy. Later she had an operation for the removal of a cyst from the liver and another for tonsillectomy. She had diseased teeth and was treated in the dental clinic. Her hearing slowly improved while she was under treatment. Dr. Dean considered her to have otosclerosis. An effort is being made to secure complete medical examinations of patients with otosclerosis.

DR. ALFRED LEWY, Chicago: Dr. Bunch's experience is similar to mine. I had a patient, a young woman, with severe anemia who could hear a whisper at 2 feet (0.6 meter). She received no treatment for otosclerosis, but therapy for anemia was instituted, and the hearing distance for a whisper was increased to 10 feet (3 meters). She had no inflammatory process in the ear. I am glad to see that the interest in the biochemical approach is gaining. Fluorine is another substance which can be used experimentally.

CORRECTION

In the article by Dr. Harry L. Baum, entitled, "A Method of Specific Treatment in Certain Streptococcic Infections," in the October issue (ARCH. OTOLARYNG. **20**:504, 1934), an error occurred on page 506. In the second line of the first paragraph under the center head "Determination of Specificity," "blood of" should be changed to "infected area in."

Book Reviews

Some Thoughts on Asthma. By A. J. D. Cameron, M.B., Ch.B., Physician to the Sherwood Park Clinic and Spa, Tunbridge Wells. With a foreword by Kenneth Playfair, M.A., M.B. Price, $2.75. Pp. 178. Baltimore: William Wood & Company, 1933.

This is a brochure of 178 pages and is what the title states—some thoughts on the subject of asthma based on the author's years of clinical experience as a practitioner of medicine. It is not a profound contribution, but it is readable. It begins with a short chapter on the nature of asthma, followed by chapters on basic toxicosis, the relations of the ethmoid sinus to asthmatic phenomena and the irritable nervous system. Then comes a long chapter on biochemistry, in which the author discusses the relation of the asthmatic symptoms to intestinal disturbances. The volume ends with a short chapter on treatment, in which emphasis is laid on general, rather than any specific, medication.

Annals of Roentgenology. A Series of Monographic Atlases. James T. Case, Editor. Volume 16. **Foreign Body in Air and Food Passages Roentgenologically Considered.** By Chevalier Jackson, M.D., and Chevalier L. Jackson, M.D. Price, $12. Pp. 265, with 236 roentgen studies and 10 clinical illustrations. New York: Paul B. Hoeber, Inc., 1934.

This volume presents a remarkable series of roentgen studies accompanied by a short text in eight chapters on the following subjects: axioms, with illustrative roentgenograms; the differential diagnosis of foreign bodies in the air and food passages; the diagnosis of radiopaque foreign bodies in the air passages; the diagnosis of radioparent foreign bodies in the bronchi; foreign bodies in the stomach and intestines; roentgenologic requirements of the bronchoscopist; collateral data useful for the roentgenologist, and fluoroscopic endoscopy. The studies will be valuable to the roentgenologist as well as to the practicing bronchoscopist.

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